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PERSPECTIVES ON ALCOHOL ABUSE

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Alcohol. A drug that can harm the body

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EDITORIAL

The "Friend" That Betrays . . .

> O God! that men should put an enemy in their mouths to steal away their brains; that we should, with joy, peasance, revel, and applause, transform ourselves into beasts.
>
> —Shakespeare (*Othello*)

People have been writing about alcohol and its effects upon the mind and body for about as long as literature as we know it has existed. For thousands of years, alcohol in one form or another has been easily available for people wishing to celebrate, to drown their sorrows, to "revel," as Shakespeare put it, or simply to socialize.

A glass of wine at dinner, a bottle of beer at the ballpark, a highball or cocktail at a party—these are innocent and time-hallowed pleasures for most people, most of the time.

But for many men and women, alcohol is a destructive force that can shatter a marriage, ruin a career, destroy one's health, and stimulate reckless behavior that results in violence and death.

Alcohol is one of mankind's oldest friends—and one of its worst enemies.

I have asked the authors of the papers presented in this special issue of *Public Health Reports* to examine the problems of alcoholism from a number of viewpoints, drawing upon the latest findings in such fields as medicine, law, psychology, and sociology.

Secretary Bowen has contributed an overview of alcoholism as a public health issue, including a status report on his 14-point program for raising public consciousness on alcoholism. That program evolved from last year's first National Conference on Alcohol Abuse and Alcoholism. (The Public Health Service (PHS) has overall responsibility for monitoring implementation of the Secretary's 14-point initiative.)

Thomas R. Burke, chief of staff for the Department of Health and Human Services, discusses the economic impact of alcohol abuse and alcoholism. The bill to American business in 1987 is estimated at $120 billion, he points out, and it is expected to reach $150 billion by 1995.

Progress in the prevention and treatment of alcoholism among the Native American population is described by Dr. Everett R. Rhoades, who heads the Indian Health Service. Dr. Frederick K. Goodwin, administrator of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA), outlines promising new lines of research, such as molecular genetics and challenge drug methodology.

Other articles in this issue examine such topics as the epidemiology of alcohol use disorders and associated disabilities, alcoholism treatment service systems, the prevention and early intervention of alcohol dependence, university student drinking patterns and problems, biological features of women's alcoholism, trends in alcohol-related morbidity and mortality, and biochemical aspects of alcohol screening.

My special assistant Dr. Sarah Moten has contributed invaluably to the production of this issue of *Public Health Reports*. Her efforts have helped to call attention to the complex of problems that relate to alcohol use and abuse.

While this special issue of the journal was being prepared, plans were being finalized for the Second National Conference on Alcohol Abuse and Alcoholism, which is being held October 30 to November 2 in San Diego, CA. The theme of this year's conference: "America Focuses on Alcohol—A National Drug Problem."

Five major areas are being explored at this year's conference. They are research; clinical issues; program administration; prevention, education, and intervention; and business and labor.

The conference is being held to encourage the development of innovative public and private solutions that address alcoholism and alcohol-related problems. Experts in the field of alcohol abuse and alcoholism, executives from business and industry, academia and health organization leaders, State and local government officials, and the general public are participating.

The message is clear and simple: We are determined to impress upon the nation the need for everyone to understand alcohol abuse and to come to grips with the many problems it creates—not only for individual alcoholics and their families but for our society as a whole.

I will not attempt to cite here all of the avenues being explored by PHS in its stepped-up war on alcohol abuse and alcoholism. But I do want to comment briefly on one aspect of Secretary Bowen's 14-point initiative—improving the clinical training of medical professionals concerning alcoholism recognition, diagnosis, intervention, and treatment—because this is a subject close to my own heart.

I feel strongly that practicing physicians should be the most knowledgeable and accessible sources of information in their communities on those medical issues that have broad social importance, such as alcoholism, problems of the aged, drug abuse, and AIDS. Whenever possible, medical people should be taking a leading role in helping to shape local responses to these issues.

Unfortunately, the average physician simply doesn't know as much as he or she should about alcoholism.

Too many doctors who otherwise are keenly alert to the most subtle clues concerning a patient's physical condition either fail to consider the possible effects of alcohol abuse on the human body or else shy away from pressing the patient concerning his or her alcohol consumption.

Even psychiatrists and other practitioners who counsel those suffering from emotional distress too often overlook or ignore the role of habitual excessive drinking.

I am told that many recovering alcoholics who rely upon the fellowship of Alcoholics Anonymous to help them maintain sobriety are scornful of the medical profession for having failed to properly diagnose their problem when they were drinking. In some cases, even when the patient wondered aloud if drinking might be a key factor in his or her situation, the suggestion was brushed aside, rather than being investigated.

This must change, and a number of approaches are being pursued to help bring about that change. They include a broad range of educational programs for physicians. Examples:

- Integrated alcohol and other drug abuse model programs for primary care physician education are being developed by seven medical schools with PHS funding. Nurse education curriculum models also are being developed.
- Conferences and workshops have been held for physicians, nurses, emergency medical personnel, hospital administrators, alcohol treatment specialists, and other medical personnel, and more conferences like those will be held.
- A quick response publication for health professionals and other interested people, "Alcohol Alert," highlights research findings in this area. The first issue was distributed by ADAMHA's National Institute on Alcohol Abuse and Alcoholism in July to more than 20,000 physicians and other health professionals.

It is vital that we update the education of the practicing physician—indeed, of the entire medical community—concerning the complex nature of alcoholism if we are to conquer this ancient problem, because the fight against alcoholism, like so many other public health issues, is best waged at the community level.

Robert E. Windom, MD
Assistant Secretary for Health

Raising Public Awareness About the Extent of Alcohol-Related Problems: an Overview

OTIS R. BOWEN, MD

Dr. Bowen is Secretary of Health and Human Services. Tearsheet requests to the News Division, Office of Public Affairs, Rm. 636, Hubert H. Humphrey Bldg., 200 Independence Ave. SW, Washington, DC 20201.

As Secretary of Health and Human Services, I have an opportunity to address a wide range of issues affecting the public health of the American people.

Few topics, however, are in as dire a need of renewed attention as are the often neglected problems of alcohol abuse and the disease of alcoholism.

Part of the problem is institutional. Moderate use of alcohol is accepted as a part of the American way of life. Yet it is a drug with the potential to do great harm, if misused in any of a variety of ways.

Another reason is the complexity of the problem. Alcohol abuse takes a toll on individuals and families, the young and the old, rich and poor. It has a bearing on both our economic health and on our competitive position internationally.

I have made it a priority of mine to do what I can to raise public awareness as to the nature and extent of alcohol abuse and alcoholism in our society. I've spent most of my life as a physician. In the field of medicine, you see plenty of pain and suffering; but you also see hope and miracles of recovery. I want to take a brief look at both sides of that coin as they relate to alcohol.

Alcohol's Physical and Societal Effects

People need to know more about the many ways in which alcohol affects the body. There's liver cirrhosis, of course—the condition most often associated with alcohol. In 1984, it was the ninth leading cause of death in the United States. But alcohol can also hurt the gastrointestinal tract, including the stomach and small intestine, affecting metabolism, blood circulation, and the absorption of nutrients, leading to malnutrition. The central nervous system, including the brain, is markedly affected by single episodes of heavy consumption, which impair driving skills and other tasks which, in themselves, require judgment and entail risk. Chronic patterns of heavy use lead to dementia and permanent loss of memory. Many people do not know that excessive alcohol use can affect the body's endocrine and reproductive systems. It can curtail testosterone levels in men and cause infertility in women.

Any drinking of alcohol by pregnant women increases the risk of malformations at birth, collectively known as fetal alcohol syndrome. An even larger number of infants suffer from long-term developmental problems because of mothers' drinking during pregnancy.

Chronic alcohol abuse means increased susceptibility to infectious diseases and cancers, in part because of alcohol's ability to interfere with the body's immune defense system.

We can scarcely speak of the immune system today without mentioning AIDS. Research is now being done on the relationship between ongoing alcohol consumption, the altered state of the immune system that can result, and the speed with which the AIDS virus can work its harm in that kind of host environment. On a related point, studies on the behavioral aspects of alcohol use have shown that sexual judgments—and that includes judgments as to both partner and practice—are often affected by alcohol. In an era when those judgments can be life or death choices, people need to be aware of alcohol's effect on decision-making.

Alcohol's Ripple Effect

While alcohol abuse breaks a person down physically, it also wreaks havoc on the family, community, health care providers, and business and industry.

Nearly 5 million adolescents, or 3 of every 10, have problems with alcohol today. It is especially disturbing to me that alcohol use by young people has been found to be a "gateway" drug preceding other drug use. Also, millions of children have a genetic predisposition to alcoholism; about 1 of every 15 kids will eventually become an alcoholic. Experts say about 4 in 10 family court cases

'The overall economic costs of alcohol-related problems are staggering. It is estimated that in 1983 alone, when one considers the health care bill, loss of productivity, and all the other factors—alcohol cost our society $117 billion. By contrast, the estimated costs of illicit drug use and abuse that year were $60 billion.'

involve alcohol. Excessive consumption is known to be a dominating factor in some one-fourth to one half of marital violence cases and one-third of child molestation cases.

We know that alcohol-related traffic deaths continue to maim and kill thousands of teens each year. In three recent studies, 75 to 80 percent of boating accidents and deaths were found to be alcohol-related and, of the fatalities recorded, more than a third of the victims were legally drunk. Likewise, a shocking number of homicides and suicides, railroad and aviation accidents, fires and falls can similarly be traced back to the victims' involvement with alcohol.

Alcohol-related problems are also putting a strain on our health care systems. One prominent physician wrote recently that at least 60 percent of all emergency room admissions in New York City hospitals are alcohol-related.

The overall economic costs of alcohol-related problems are staggering. It is estimated that in 1983 alone, when one considers the health care bill, loss of productivity, and all the other factors—alcohol cost our society $117 billion. By contrast, the estimated costs of illicit drug use and abuse that year were $60 billion. What is more, the $117 billion aggregate cost is projected to rise—to $136 billion by 1990 and to $150 billion in 1995.

I believe that, by working together to address alcohol-related problems, we can help to make life better for individuals, families, and entire communities. People who hear the messages about alcohol and understand and act upon them can be healthier and more productive in their jobs. Families would experience fewer domestic problems, and health care expenses for them would slack off. Other social problems would diminish, easing the strain on community and government resources. But we will only turn the corner if each of us takes an active role in learning, sharing information, and committing ourselves to solve these problems in our daily lives.

I mentioned earlier the sense of hope and the miracle of recovery that motivate strongly many in the medical professions. And certainly, there's reason for real hope amidst the clouds of pain and suffering for those either directly or indirectly suffering from alcohol-related problems today. Medical research has gone a long way to help us better understand alcohol abuse and alcoholism. Prevention and treatment programs are each day helping more Americans halt the onset of alcohol-related problems or rescue them once they've begun experiencing problems. Research, prevention, early detection, and treatment are the greatest forces operating today for controlling and reducing alcoholism.

Thanks to the efforts of concerned family members, health care professionals, educators, clergy, the media, and others, the stigma affecting those who abuse alcohol and suffer from alcoholism is being lifted. Though many still hurt behind closed doors, people with alcohol problems today are hiding less and communicating more. Silence is each day giving way to courage—and shame, to strength. The pain still remains for many, yet there's also so much potential for hope.

That is why in May of 1987, I chose a Planning Committee of distinguished persons from the alcohol field and asked them to help me determine what needed to be done to make people aware of the progress thus far, as well as what work remains. These top professionals and dedicated volunteers welcomed the challenge I gave them and formed into several different subcommittees to go about their work.

I reviewed the exhaustive reports they prepared for me after several months of hard work. And then, in November, last year, Mrs. Betty Ford and I co-chaired the first ever Secretary's National Conference on Alcohol Abuse and Alcoholism. There, I set forth a comprehensive 14-point plan to raise public consciousness about alcohol-related issues.

Let me give a brief status report on each point.

Coordinating HHS Responses

First, with the goal of increasing the emphasis on alcohol-related problems and better coordinating our Federal response, I've directed my Assistant

Secretaries to summarize all their ongoing alcohol-related activities for last year and identify those planned for this year and next. The National Institute on Alcohol Abuse and Alcoholism (NIAAA) has had these survey results analyzed and a report prepared. Alcohol abuse and alcoholism are intrinsic to many problems addressed by my Department—including health care financing, family support, hypertension, heart disease, and cancer. We must support research and a sharing of information so that more people learn about these links.

Second, my Assistant Secretary for Public Affairs is currently surveying public affairs officers throughout the Department of Health and Human Services (HHS) to find new ways to cite alcohol research findings in their work with the media and other outside organizations.

Toward this same end, I've directed the Public Health Service (PHS) to develop a quick response publication to provide timely information to physicians and others on alcohol abuse and alcoholism. The first two issues of "Alcohol Alert"—one on treating methadone-maintained patients in alcoholism treatment programs and the second on alcohol and aging—are now available. Distribution is being handled by our National Clearinghouse for Alcohol and Drug Information.

At my request, NIAAA officials have met with the American Association of Medical Colleges and the American Nurses Association to discuss how research findings can be used in medical and other education programs. Our goal is to improve training in alcoholism recognition, diagnosis, intervention, and treatment. We have also held a workshop on curriculum changes and licensure issues.

Third, with help from the National Council on Alcoholism, I've established a privately run National Citizens Commission on Alcohol Abuse and Alcoholism. Surgeon General C. Everett Koop has agreed to serve as a Trustee of the Commission. By encouraging high-visibility events such as public testimonials by prominent leaders and by taking on any number of special projects, the new Commission can have a lasting impact on community awareness and further reduce the stigma of alcohol-related problems. Its Executive Committee met on July 19 and the full Commission held its first meeting on August 10, 1988.

Stepping Up Educational Programs

Fourth, though we do not have regulatory jurisdiction over health warning labels at HHS, we've studied their effectiveness at the request of Congress and provided our report on the health hazards associated with alcohol use. We made no specific recommendations, but we did cite the effectiveness of health warning labels in other areas—like tobacco and food and drugs. The Treasury Department, which has jurisdiction, has also been requested to study this, and I am waiting to see their report before taking any further action on this matter.

Fifth, I'm directing my Assistant Secretary for Public Affairs to see how we might best work with the public and private sectors to secure voluntary changes in radio and television programming to promote the avoidance of alcohol misuse by young people or other persons at high risk. The staff has met with the Federal Communications Commission and the Federal Trade Commission and will be working with private broadcast groups as well.

Our sixth initiative has to do with assessing alcohol and other drug education and prevention programs.

I believe we must encourage innovative, community-based programs to prevent alcohol abuse, and our newly created Office for Substance Abuse Prevention has lead responsibility for this. The "Be Smart, Don't Start—Just Say No" campaign to prevent alcohol use by 8- to 12-year-olds is a good example of how I believe Federal efforts can and should supplement efforts at the State and local levels and by the private sector.

Knowing that research on the effectiveness of alcohol education programs is limited, I have directed the Assistant Secretary for Health to evaluate existing programs and look at new approaches to reduce alcohol use by youth. One component of this evaluation will be a survey of alcohol and other drug prevention curriculums in kindergarten through grade 12. I have also asked the PHS to see to it that all HHS activities, reports, and other documents that relate to preventing alcohol and other drug abuse pay specific attention to preventing alcohol abuse and alcoholism, commensurate with the impact of these problems on our nation.

Early in this initiative, I asked William Bennett, former Secretary of Education, to pay special attention to the alcohol problem when developing and implementing alcohol and other drug abuse prevention programs in his department. He was especially cooperative, working with us to distribute a curriculum guide on alcohol prevention to some 47,000 elementary schools across the United States.

'According to the Wall Street Journal, brewers and beer distributors spend $15–$20 million each year in campus marketing. Alcohol use on college campuses is pervasive, contributing to poor grades, excessive vandalism, many injuries and, not so infrequently, death. Several States have restricted alcohol promotions on campuses. . .'

Seventh, I've directed my Assistant Secretary for Public Affairs to develop a comprehensive public affairs plan to highlight alcohol abuse and alcoholism. This multifaceted plan capitalizes on the expertise of the National Association of Broadcasters and the respect accorded role models such as Bill Cosby and 1984 Olympic gold medalist Nancy Hogshead. It also uses the knowledge of nationally renowned experts in the field of alcoholism. In addition, I've participated personally in this plan by recording several alcohol-related radio spots as a part of our "Housecalls" series of public service announcements.

Alcohol-Related Health Benefits and DRGs

Eighth, I know that employee assistance programs play major roles in early intervention and referral to treatment for employed alcohol abusers and alcoholics. Small businesses represent 65 percent of our work force, yet studies show that only 25 percent of our work force has access to such programs. That's why I have written to the Secretary of Labor asking that the Labor Department join us in notifying small businesses of the potential that employee assistance programs and alcohol-related health benefits possess as effective cost-containment tools. We have since met with the Departments of Commerce and Labor, Small Business Administration, National Federation of Independent Businesses, and other private sector groups to determine how best to reach small businesses.

Next, the National Council on Alcoholism has estimated that 5 percent of the Federal work force suffers from alcoholism. The implications of this estimate on the productivity of a 3 million-member work force are deeply troubling.

Last year, the Office of Personnel Management (OPM) successfully negotiated with major Federal health care insurers for the coverage of alcohol and other drug abuse treatment in policies available to Federal employees. I've expressed my thanks to the director of OPM for their role in this development and requested that Federal employees be made more aware of the potential productivity losses due to alcohol and of the help available.

Tenth, we'll continue, under my watch as Secretary, to pay close attention to the many factors affecting Medicare and Medicaid reimbursement for alcoholism treatment. On September 1, 1987, we issued final regulations making changes to Medicare's diagnosis-related groups (DRGs) and payment rates for FY 1988. These regulations established a number of new DRGs for alcohol and drug abuse and terminated the exclusion for specialized alcohol hospitals and units. Our experts believe these changes will result in equitable payments for alcohol-related services to Medicare beneficiaries and their families. Since research is continuing on these Federal payment issues, I've directed our financing experts to keep my office informed of any special new developments on the Medicare or Medicaid fronts.

Treatment for Families of Alcoholics

Eleventh, although attention is often paid to the need for treating abusers and alcoholics, many people do not know that families of those with alcohol problems need counseling as well. Nor is it very well known that many alcohol treatment programs offer special family services.

I have written the Assistant to the President for Policy Development, Gary Bauer, requesting his assistance in alerting Americans to the impact of alcohol abuse and alcoholism on families and to the fact that treatment is available and effective for families of alcoholics. He has graciously agreed to help. In addition, I'll be surveying our program administrators to identify all ongoing Federal efforts to serve families of alcoholics and to identify and encourage new activities planned for this year and next.

Twelfth, many people don't know that alcohol, even in small amounts, can affect coordination and performance. Yet some alcohol advertisements depict people in high-risk activities—for example, kayaking and swimming—which could be life-threatening if done while using alcohol. Hopefully, we will be able to work with the private sector to alert people to these potential dangers.

Alcohol Use on College Campuses

Next, I want to encourage colleges and universities to be more than passive observers of the massive marketing and promotion activities designed to encourage consumption of alcoholic beverages on our nation's campuses. I recently addressed a group of college administrators on this problem. According to the Wall Street Journal, brewers and beer distributors spend $15–$20 million each year in campus marketing. Alcohol use on college campuses is pervasive, contributing to poor grades, excessive vandalism, many injuries and, not so infrequently, death.

Several States have restricted alcohol promotions on campuses and some schools have done the same on their own. A college-age youth task force has been convened and new materials went out to campuses this fall. I think that, with a continued partnership involving educators and student groups, we can stimulate States and individual colleges and universities to take a fresh look at this problem and act accordingly.

Call to Action

Finally, I agree with the many alcohol professionals who feel that the use of the broad term ''substance abuse'' diminishes public attention to alcohol-related issues, although alcohol is a major part of the overall drug problem. Therefore, I'm requesting that, from now on, all HHS components use the term ''alcohol and other drug abuse'' in all future documents, unless legislation requires otherwise.

These are the major components of our Initiative; but they're by no means the be-all and end-all of a national strategy to battle alcohol-related problems. Rather, I hope all this is just a beginning. I want to invoke a new understanding of problems that have long plagued our nation. To do anything less would be a disservice to ourselves, our society, and to the many future generations whose lives and livelihoods are at stake. Although significant progress is being made, more people need to know that we can prevent alcohol abuse in America and that alcoholism is a disease, and this disease is highly treatable.

My aim in this article is to remind public health professionals of the magnitude of these problems—and to challenge you to take every opportunity that comes your way to prevent alcohol problems from further crippling our health and social systems and our economy.

In the broader sense, I've invited Americans from all walks of life—business, media, government leaders at all levels, educators, civic groups, clergy, and respected figures in sports and entertainment—not just to think about these problems in new ways, but to act in new ways as well.

Only together will we make new and lasting progress.

The more you raise your glass, the more you may be raising your blood pressure.

If you have high blood pressure,
your blood pressure can go up with the
amount of alcohol you consume.

So remember, take it easy on the alcohol. . .
if you want your high blood pressure
to take it easy on you.

Treat your
HIGH BLOOD PRESSURE.
Treat yourself right.

The National High Blood Pressure Education Program, The National Heart, Lung, and Blood Institute, National Institutes of Health, Public Health Service, U.S. Department of Health and Human Services

The Economic Impact of Alcohol Abuse and Alcoholism

THOMAS R. BURKE

Mr. Burke is Chief of Staff, Office of the Secretary, Department of Health and Human Services.

Tearsheet requests to Mr. Thomas R. Burke, Office of the Secretary, Room 606G, Hubert H. Humphrey Building, Washington, DC 20201.

Synopsis

The economic effects of alcohol abuse are as damaging to the nation as the health effects, affecting the family, the community, and persons of all ages. Underaged drinking is interfering with children's development, affecting the nation's ability to respond to economic challenge in the future. The college aged may be the most difficult to educate about alcohol abuse because of drinking patterns established at an early age and susceptibility to advertising inducements.

Health care costs for families with an alcoholic member are twice those for families without one, and up to half of all emergency room admissions are alcohol related. Fetal alcohol syndrome is one of the top three known causes of birth defects, and is totally preventable. Alcohol abuse and alcoholism are estimated to have cost the nation $117 billion in 1983, while nonalcoholic drug abuse that year cost $60 billion. Costs of alcohol abuse are expected to be $136 billion a year by 1990, mostly from lost productivity and employment. Between 6 and 7 million workers are alcoholic, with an undetermined loss of productivity, profits, and competitiveness of American business. Alcohol abuse contributes to the high health care costs of the elderly beneficiaries of Federal health financing programs. Heavily affected minorities include blacks, Hispanics, and Native Americans.

Society tends to treat the medical and social consequences of alcohol abuse, rather than its causes. Although our experience with the consequences of alcohol abuse is greater than that for any other drug, public concern for its prevention and treatment is less than for other major illnesses or abuse of other drugs. Alcohol abuse is a problem being given high priority within the Department in an effort to create a national agenda on the issue and to try to impart a greater sense of urgency about the problems. Ways are being explored to integrate alcoholism activities into more Departmental programs. Employee assistance programs for alcohol abuse have been established in about 90 percent of major companies, and Federal employees' access to treatment is being expanded through health insurance coverage. Collective public efforts are required to encourage prevention and treatment efforts, and to begin to reduce the economic effects of alcohol abuse that the country can no longer afford.

ALCOHOL is the foremost drug of abuse in the United States today. The consequences of alcohol abuse, including the disease of alcoholism, exact a far greater toll on our health and on our social well-being than the abuse of any other substance. Because of my training as an economist, I tend to evaluate the economic aspect of issues. In my view, the effects of alcohol abuse and alcoholism are as damaging to our nation's economy as they are to our nation's health.

No single segment of our society is untouched by this phenomenon—not the young, not the middle-aged, not the elderly. Alcohol abuse affects the entire family; it troubles every community. Time and again, we have seen how it tears at the very fabric of American life.

Studies show that the consumption of alcohol can begin very early. It is not uncommon these days for children to begin drinking before they have reached the age of 12 years. Elsewhere in this journal, medical experts attest to the harm that alcohol can do to young minds and bodies—indeed, to the emotional and physical health of people of any age.

Let's look, again, at the children who are drinking, including those who are drinking wine coolers when their beverage of choice ought to be fruit juices, milk, and soft drinks.

Underage Drinking

Children of any age—from toddlers to teenagers—should not have their minds clouded by alcohol—or any other drug for that matter.

Our children are our hope for the future; it is they who are going to keep our country strong. If we are to maintain our high wage-based economy, today's children are going to have to be prepared to face some of the most difficult challenges in the future. We currently compete in a tough international marketplace for goods and services. That marketplace will become even more competitive as the global market expands. As this new generation reaches adulthood, it will need to have more flexible skills and an increased ability to sustain peak performance levels in order to roll with the high technology punches and to successfully compete in the international marketplace with countries like Japan. This was not true with earlier generations, raised during and immediately after WW II, when the United States was the most highly industrialized nation in the world, and international competition was virtually nonexistent.

Another stratum of our society that concerns us greatly lies between the high-school-age population and the adult working-aged population. I refer, of course, to the nation's college students. I suspect that they may be the most difficult group to reach with the facts about alcohol abuse. They are important because it is quite literally true that they will be tomorrow's leaders. They can be difficult to reach, or to educate, because they are at a time of great change in their lives. While often viewed as adults by society, they are not always equipped, when they reach the minimum drinking age, to make informed choices about drinking. Too often, they are easy prey for the beer, wine and liquor industries, with their slick marketing programs and skillful advertising techniques, such as promotional nights, drinking contests, and sports and club sponsorships. In addition they have established drinking patterns and attitudes from their early teens which may be difficult to extinguish.

In this connection, athletic participation is an important part of growing up for millions of young men and women in this country. But 50 percent of all high school coaches surveyed in a recent USA Today poll said that alcohol use by their young athletes is very serious and getting worse. The most often cited causes of the problem were social acceptance of alcohol, a glut of TV advertising, and the ease with which alcohol can be purchased. Drinking is made to seem like a "glamorous and grownup thing to do," one coach remarked.

It is small wonder, when you stop to think about it, that some 4.6 million adolescents in America experience serious alcohol problems each year. And should we be surprised to learn that more than 1 out of 2 teenage deaths in auto accidents involve the use of alcohol?

Direct Costs

In 1983, health care costs alone for illness and trauma related to alcohol abuse totaled $15 billion. Studies show that the average monthly health care bill for families in which there is an alcoholic member is twice that of families without an alcoholic member. It is estimated that 20 to 40 percent of all inpatient hospital admissions and up to 50 percent of all emergency room admissions are alcohol related. On the brighter side, there is considerable evidence that treating alcohol-related problems reduces expenditures for overall health care costs, not only for the alcohol abusers and alcoholics, but for their families, as well.

While the illness costs figures are shocking, even sadder is the fact that alcohol abuse and alcoholism destroy families, the very foundation of our society. They work their destruction in many ways. Alcohol is involved in one-quarter to one-half of all marital violence cases, in one-third of all child molestation cases and in about 13 percent of child abuse cases reported to police.

To talk about families is to talk about pregnancy and birth. Studies show that for one-half of all teenage pregnancies, one or both parents had been drinking alcohol at the time of conception. Drinking while pregnant makes a prospective mother much less likely to give birth to a healthy baby at full term, yet many mothers-to-be continue to drink (and smoke, unfortunately) during their pregnancies. In so doing, they are taking a real chance that their baby will be born with physical, mental, or behavioral abnormalities or with fetal alcohol syndrome. Fetal alcohol syndrome is one of the top three known causes of birth defects in the United States today, and the only one that is totally preventable.

Indirect Costs

We have looked at some of the direct costs of alcoholism and alcohol abuse, with particular reference to children and young people. Many experts say that the direct costs pale when compared to the indirect effects and costs to society. Consider the use and abuse of alcohol in the working-aged population, where the costs are enormous but more difficult to break out.

> *'No single element of our society is untouched by this phenomenon—not the young, not the middle-aged, not the elderly. Alcohol abuse affects the entire family; it troubles every community.'*

It was estimated in 1983 that alcohol abuse and alcoholism were costing our economy $117 billion a year. In comparison, nonalcoholic drug abuse that year cost $60 billion. Costs of alcohol abuse are projected to rise to $136 billion by 1990 and to $150 billion in 1995.

Of the $117 billion, the greatest portion—$92.8 billion—came from reduced worker productivity and lost employment. Put another way, $92.8 billion was the price tag for the products, goods, and services that were never produced or delivered that year because alcoholic workers were less productive, because they lost their lives prematurely to alcohol-induced illness and accidents, and because society incurred the costs of incarcerating so many of them for their criminal activities.

At present, between 6 and 7 million employed American workers are alcoholics. Their affliction affects American business adversely, first and foremost in the form of increased production cost and decreased profits and, second, in its effect on the competitiveness of American business in the international marketplace.

Everyone is worried about the foreign trade deficit. Have you ever heard alcohol abuse or alcoholism mentioned in connection with this problem? Probably not. Yet many of the goods produced in this country are defective from the start, and need replacement or fixing. The result, of course, is increased prices, which reduce the attractiveness of our products on the world market and, ultimately, cut profits and jobs from the economy. Alcohol is a productivity problem we can't afford.

The situation in the Federal Government is no different. Productivity is the concern, and alcohol abuse and alcoholism have a direct and substantial impact on it.

Most of us are at least passingly familiar with the medical consequences of alcohol abuse—heart and other cardiovascular diseases, hypertension, cirrhosis of the liver, and cancer, and the list goes on. For an elderly person on medication for treatment of these and other illnesses, the combination of alcohol and medication can render the medication ineffective or lethal.

Alcohol abuse and alcoholism hit hard in the elderly segment of the population, contributing to extraordinarily high health care expenditures. These costs are paid by you and by me and by every other taxpayer in the United States through Federal health financing programs such as Medicare and Medicaid. Suffice it to say that the drain on the Federal Treasury for these and other government financing mechanisms, such as funds for the homeless and family support programs, is enormous.

A recent study estimated that as many as one-half of all our homeless in America suffer from alcohol abuse and alcoholism. Alcohol is one of the chief causes of illness and death among Black, Hispanic, and Native American populations. The incidence of alcohol-related medical problems, particularly liver cirrhosis and cancer of the esophagus, is very high among blacks. Cirrhosis mortality rates for blacks are twice as high as the rates for whites. Even though many tribes remain almost totally abstinent, alcohol-related illness and injury among American Indians is three times the rate of the general population. Accidents, most of them alcohol-related, are the second most common cause of death and account for nearly one-fourth of deaths among American Indian men. Hispanic men in this country have a higher rate of alcohol use and abuse and a higher rate of cirrhosis mortality.

Better Health and Reduced Costs

I share with Secretary Bowen the view that we, as a society, could ameliorate many of our health care problems by preventing and treating alcohol abuse and alcoholism. I believe, too, that if we could just get a handle on our alcohol problem we could, in addition to improving the health and quality of life of millions of Americans, cut our expenditures for health care dramatically.

An amazing phenomenon, in view of the ever-escalating costs of health care and welfare, is that we continue to treat the medical and social consequences of alcohol abuse and alcoholism, rather than the cause. We focus on the homelessness of street people, on their lack of shelter and food and warm clothing. For nearly half of them, homelessness is just a symptom; the disease is alcoholism.

Our experience with the consequences of alcohol abuse in America is greater by far than our experience with any other drug. Yet, we have not seen the kind of public concern over the problem and public pressure to prevent it and treat it that we have seen with other major illnesses or the abuse of other drugs. Part of the explanation may have to do with the fact that alcohol is a legal drug. But so is tobacco, and we've made great progress in reducing its use. Indeed, people today are more aware of the health effects of passive smoking than they are of abusing alcohol, even though alcohol abuse is a far graver problem.

We hear and read daily about the threat that illicit drugs pose to our nation. Without meaning to belittle that threat in any way, for it is real, it is serious, and it is deserving of all the efforts that are being made to overcome it. I note that the costs of alcohol abuse and alcoholism, which amounted to roughly $120 billion last year, far exceeded the costs of all other drugs of abuse combined.

A Top HHS Priority

We need to raise the visibility of alcohol abuse and alcoholism in our nation, just as we have for the hazards of smoking. Dr. Bowen has a longstanding interest in the problems of alcohol abuse and alcoholism. He is very concerned about the apparent lack of attention the public pays them. That is one of the chief reasons for his announcement, early last year, that for the remainder of his term as the Secretary of Health and Human Services, he is making alcohol abuse and alcoholism one of his top priorities. His aim, he made clear, is to use the high visibility of his office to create a national agenda for the prevention and reduction of alcohol abuse and alcoholism, and to impart to the American public a sense of urgency about these problems.

There are programs in HHS that focus some of their efforts (or all their efforts, in the case of the National Institute of Alcohol Abuse and Alcoholism) on alcohol-related problems. We are now exploring ways of integrating alcohol abuse and alcoholism activities into the on-going work of many other HHS programs whose missions are naturally compatible with the objectives of our alcohol initiative.

We are looking for ways to use public education more effectively, perhaps by employing as national spokespersons nationally prominent persons who are recovering alcoholics, and by obtaining major public service advertising and the help of professional advertisers for alcohol-related media campaigns. Our "Be Smart! Don't start!—Just Say No!" campaign is an example of this kind of activity.

We are exploring ways to counter the impact of alcoholic beverage advertising and promotion, particularly those campaigns that fail to discourage underage drinking, or drinking by individuals who are at high risk for negative health consequences, such as pregnant women and persons who should not drink because of medications they are taking.

Our concern, at base, is that TV commercials that "sell" alcoholic beverages to viewers of all ages—and perhaps especially commercials that use well-known sports figures and other celebrities—are slick, pervasive, and persuasive. We believe we need some countervailing forces, some equal time on TV, to show the other side of the story.

This summer and into the fall, HHS is sponsoring a number of alcohol research and treatment conferences and symposia. One that deserves mention is a workshop, sponsored by NIAAA and the National Center for Health Services Research, the purpose of which is to develop an agenda for health research economics related to preventing and treating alcohol-related problems.

Employee Assistance Programs

This year, some 2.2 million Federal employees will have access to alcohol treatment, if they need it, as a result of expanded insurance coverage in their employee benefit plans. We worked with the Office of Personnel Management to make this health insurance coverage available, and we are assisting in publicizing its existence throughout government. As we have seen, alcoholism has direct and substantial consequences for the productivity of any organization, including the Federal Government. We also know that the use and cost of health care services decline once treatment for alcoholism is begun.

We are taking these facts and the lessons we have learned with the Federal work force to the private sector. Lest this assertion sound presumptuous, I should note that roughly 90 percent of the Fortune 500 companies already have established employee assistance programs (EAPs). Our main target is the nation's small businesses—those that employ 250 persons or less and that, by and large, have not established EAPs. Because small businesses comprise approximately 65 percent of the nation's work force, reaching employers in this segment of the working population is clearly essential if we are to stem the growing tide of alcohol abuse in America and reclaim the billions of dollars in worker productivity now lost to the effects of alcohol abuse and alcoholism.

'At present, between 6 and 7 million employed American workers are alcoholics. Their affliction affects American business adversely, first and foremost in the form of increased production costs and decreased profits and, second, in its effect on the competitiveness of American business in the international market place.'

Conclusion

Alcohol abuse is a problem that affects us all. It is a national health problem of the highest order. In recent years, Americans have gained a better understanding of the complexity of our alcohol problems, but they still do not know all they should. Many just don't realize how extensive alcohol abuse and alcoholism are, and how these problems affect not just individuals, but families, schools, workplaces, health care systems, and, ultimately, our entire economy.

I believe that our Department—in particular, the National Institute of Alcohol Abuse and Alcoholism of the Public Health Service—has done a fine job of accumulating knowledge about alcoholism and of making this knowledge available to the public. I believe, too, however—indeed, I know for a fact—that we must do more.

Last year, as I noted, America's bill for alcohol abuse and alcoholism came to more than $120 billion. Forecasters familiar with this country's alcohol problem have predicted that these costs will increase to $136 billion in a little over a year from now and to $150 billion by 1995.

Are these increases inevitable? The question is not easily answered, but my own view is optimistic. I believe we can turn this trend around, and someday prove the forecasters wrong, if we work hard and together to raise public awareness of the costs and consequences of our alcohol-related problems and if we channel this awareness, this heightened concern, into programs of research, prevention, and treatment that leave no room for anyone to doubt the seriousness of our intentions. Our economy simply cannot continue to absorb an annual expense that runs to 12 digits, is on the rise, and is largely preventable. Alcohol abuse and alcoholism, we must all come to understand, are problems we can no longer afford, or ignore. The time for effective intervention is now.

Bring Yourself Up To Date

Alcohol and Birth Defects: The Fetal Alcohol Syndrome and Related Disorders

A new booklet from NIAAA reviews advances in the understanding of the effects of maternal alcohol consumption on the unborn child, and addresses such questions as:

How prevalent are FAS and less severe effects of alcohol consumption by pregnant women?

What risks to the fetus are involved across the full range of possible maternal drinking levels?

What are the underlying mechanisms of FAS and Fetal Alcohol Effects (FAE)?

Are there critical times during pregnancy when the risk is greatest?

What are the most effective strategies for preventing FAS and FAE?

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Alcoholism Research: Delivering on the Promise

FREDERICK K. GOODWIN, MD

Dr. Goodwin is the Administrator of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA). Before his appointment to that position in July 1988, he was the Director of Intramural Research at the National Institute of Mental Health. Tearsheet requests should be sent to him at ADAMHA, Rm. 12-105 Parklawn Bldg., 5600 Fishers Lane, Rockville, MD 20857.

Synopsis ....

Prospects for research advances in alcoholism are very promising, because of the explosion in the neurosciences and advances in epidemiology and typology of the disorder. For example, the field is now ready for molecular genetics studies of the early onset form of alcoholism that is transmitted from father to son with high penetrance. Leading neuroscientists are being recruited into alcoholism research.

Paradoxically, this time of new hope coincides with challenges to the scientific enterprise, such as the animal rights movement and impatience with the scientific process in the face of the public health emergencies represented by acquired immunodeficiency syndrome (AIDS) and drug abuse.

The emergence of genetically based subtypes of alcoholism suggests that at least two discrete illness processes are involved. Mounting evidence from spinal fluid studies has rekindled interest in a key role for serotonin in the early onset form of alcoholism. One hypothesis now being explored is that genetically low brain serotonin function may be part of the predisposition to this form of alcoholism. It is known that acute alcohol intake transiently increases brain serotonin turnover. Thus, drinking might be viewed as an attempt to correct a deficit, only to produce further serotonin depletion as the drug's effect wears off, setting up a vicious cycle of repeated attempts to self-medicate.

Impulsive, violent, and suicidal behavior as well as alcohol abuse are associated with the low brain serotonin activity. Persons with these problems suffer from circadian rhythm and glucose metabolism disturbances that may also be mediated by serotonin. New pharmacological probes are now available to tease out the mechanisms of altered serotonin function.

The progressively deteriorating course of severe episodic alcoholism in many ways parallels the process of electrically kindled seizures in experimental animals. There is evidence that repeated withdrawal episodes may kindle a worsening course, including phobic disorders, perhaps by triggering a hyper-reactive noradrenalin system.

As I HAVE just recently assumed the responsibilities of Administrator of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) at this writing, what follows should be taken as a personal statement about my thinking with respect to research prospects in alcoholism. I still have much to learn about developments in this field and am looking forward to working with Dr. Enoch Gordis, Director of the National Institute on Alcohol Abuse and Alcoholism (NIAAA), and his able staff to expand my horizons during the coming months. By making the most of the exciting new research opportunities that are arising, we can deliver on the promise of new hope for persons suffering from this major public health problem.

My own occasional contributions to the alcoholism research literature have stemmed from an interest in the neurobiology of affective and impulse control disorders, and in the intersection between the two. Over the years, I have helped to train younger investigators who are now working in the alcoholism field, among them Dr. Markku Linnoila, Director of NIAAA's Laboratory of Clinical Studies. At the National Institute of Mental Health (NIMH), we have also nurtured lines of research that have potential importance for alcoholism treatment, such as Dr. Steven Paul's explanation of how alcohol's anti-anxiety effects may be mediated through a receptor complex closely linked to that which mediates the action of benzodiazepines. In working with patients, I have come to respect the powerful negative impact that even

'Simply delivering more services when we are not yet sure which interventions work best, and under what circumstances, may not be in the best interest of the public health—especially if it means curtailing research investments that could lead to the development of better treatments in the long run.'

moderate drinking can have on the course and treatment response of patients with recurrent affective disorder. In this regard, I have become increasingly impressed with the essential role that Alcoholics Anonymous and other self-help and group therapy programs play in the recovery process for persons with this often intractable illness. Through my wife Rosemary, a clinical social worker in the Substance Abuse Clinic at Georgetown University School of Medicine, I have gained a new respect for both the devastation wreaked by alcohol on young lives and for what can be done through a vigorous, comprehensive treatment program.

The Promise

We are entering an extraordinarily promising era for research advances in alcoholism. The new opportunities are based on the expansion of our knowledge base in the neurosciences—molecular genetics, molecular biology, neuropharmacology, and brain imaging techniques. As in other clinical fields, the hoped-for progress will depend on fundamental developments in basic research. Although one cannot always predict which clinical problems are going to benefit first from such advances in the basic infrastructure, the potential benefits warrant investment in broad-ranging studies.

For example, the field of molecular genetics is rapidly moving toward the mapping of the entire human genome. Alcoholism represents one of the ripest areas for clinical exploitation of these powerful methodologies, particularly early onset alcoholism, which is transmitted from father to son with high penetrance. The epidemiologic base needed to undertake the necessary clinical studies is now in place. It was such an epidemiologic base that made possible the recent discovery that a gene exists on the short arm of chromosome 11 that predisposes for manic depressive illness among the Pennsylvania Amish (*1*). Another gene locus linked to the disorder has been found on the X chromosome in a group of Israeli families (*2*). While the alcohol field hasn't yet reached this stage, it is similarly poised. All of the indirect data—family history, identical twin and adoption studies, biological markers of vulnerable children—point to the likelihood that a gene or genes linked to the early onset form of alcoholism can eventually be found.

When such genes are discovered, the awesome power of molecular genetics techniques comes into its own. At a minimum, linked genes can be used as markers for early detection, counseling, and preventive interventions. Genes can be sequenced, cloned, and expressed to discover the proteins they code for. Genes offer shortcuts to understanding and treating the underlying illness process, which may turn out to involve defective enzymes, transmitters, receptors, or other neuronal components. We already have some clues about what some of these components might be. As I will discuss later, emerging evidence linking early onset alcoholism to alterations in brain serotonin systems may be among the leads worth following up in the coming years.

When top scientists from cutting-edge disciplines become interested in an area, it is a sure sign that the field is ripe for discovery. It is no coincidence that neuroscience leaders, such as Dr. Floyd Bloom of the Scripps Institute, have been recruited into alcoholism research in recent years.

A Paradox

Paradoxically, just as a return on our investment in science seems within our grasp, political threats to the research enterprise itself have surfaced. Foremost among these is the radical animal rights movement, which has targeted precisely the kind of biobehavioral research that the ADAMHA institutes must continue to invest in if we are to carry out our mission. I speak here not about the animal welfare community, with whom we share a common concern for the well being of research animals, but about the extremists who would stop all animal research in the name of a misguided sentimentalism.

Unfortunately, rather than attempting to convince the public of their belief that animals and humans are morally equivalent, many animal rights activists seem to have chosen to exploit the public's lack of knowledge about biobehavioral disorders and the nature of research. They implicitly play upon outdated notions that mental illness and substance abuse somehow stem from uniquely human failings. It is now clear that these serious health problems involve brain mechanisms gone awry, mechanisms that are potentially fathomable, in part, via studies in animals with which we share many biological and behavioral characteristics. Animal rights proponents ignore or seriously distort the scientifically driven medical advances of the past quarter century and appear to discount the possibility of major new discoveries.

Of course, this does not mean that we give scientists carte blanche to use animals frivolously. Animals involved in ADAMHA-supported studies are cared for in the most humane manner possible. Cruelty to any animal is reprehensible and will never, under any circumstances, be permitted. Not only would it be inhumane to the animal subject, it would also be bad science, since any data obtained (especially in behavioral studies) could be confounded by the distress of the animal. By far the great majority of animal studies—90 percent—are conducted in rodents and other lower animals (*3*). For every dog or cat that serves humanity in research, 50 are killed at animal shelters (*4*). Primates are used only when the organ system in other species differs too much from that of humans.

If research on animals were halted, clinical research on brain illnesses would soon be crippled and eventually die. Though animal studies constitute only one-fifth of the ADAMHA research effort, they are the foundation for the entire enterprise. Cut the roots and the tree will die and, with it, its fruits. The major organizations representing persons ill with alcoholism have joined with their counterparts representing other illnesses and the health professional community in voicing support for continued use of animals in research.

Yet another challenge to research comes from political pressure to solve problems quickly. It is the responsibility of our scientific leadership to explain to our political leaders why we can only push science so fast. Sometimes short-term solutions to problems may damage the acquisition of long-term knowledge upon which real solutions ultimately depend. Simply delivering more services when we are not yet sure which interventions work best, and under what circumstances, may not be in the best interest of the public health—especially if it means curtailing research investments that could lead to the development of better treatments in the long run. For example, during the polio epidemic of the early 1950s, there were very strong pressures to spend large sums to perfect portable iron lungs. In retrospect, it turned out to have been a very wise decision to instead invest the bulk of March of Dimes money in basic biological research.

Today, we face similarly pivotal issues in substance abuse research policy. In some quarters, our research effort is branded "ivory tower," unresponsive to pressing public health needs. In fact, we really do have information gaps. We need to close them as quickly as we can in the face of a public health emergency. Yet, as we have seen with AIDS, even when we expedite a promising new discovery from the test tube into clinical trials, it still may seem too slow to people who do not understand the scientific process.

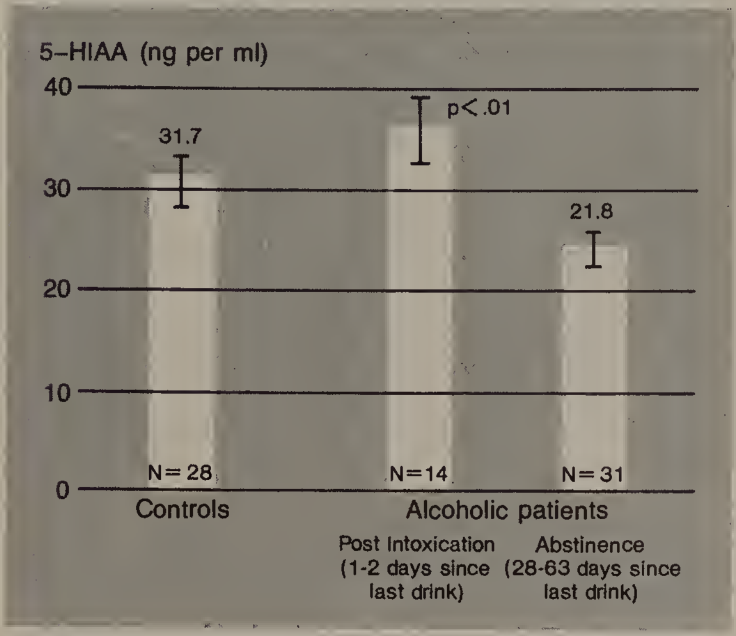
Serotonin Hypothesis Revisited

One outgrowth of my early research on the pathophysiology of depression has been an interest in the relationship between the neurotransmitter serotonin and impulse control problems. A hypothesis I helped to formulate a decade ago concerning a role for serotonin in alcoholism continues to influence some clinical investigations. In the late 1970s, our group at NIMH (*5*) assessed brain function and personality characteristics of sailors in treatment for alcoholism at the National Naval Medical Center. Spinal fluid samples were assayed for levels of 5-HIAA (5–Hydroxyindoleacetic acid), a byproduct of the metabolic breakdown of serotonin that provides an indirect window into turnover of the neurotransmitter in brain. We found that upon admission when still intoxicated, the alcoholic sailors' 5–HIAA levels were relatively higher than a control population's. After 4 weeks of abstinence, their 5–HIAA levels had dropped significantly below those of nonalcoholic controls with similar personality profiles (fig. 1).

On the basis of this and animal research findings, we speculated that (*5*) (fig. 2)

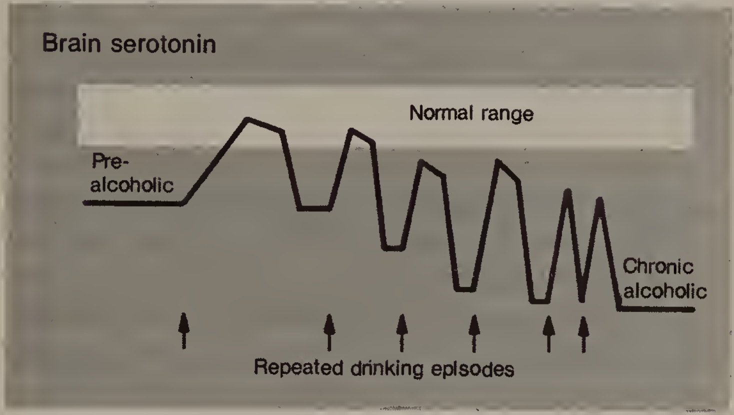
> . . . the pathophysiology of alcoholism might involve preexisting low brain serotonin levels that are increased transiently by alcohol consumption, but that brain serotonin levels gradually undergo increments of further depletion as a consequence of repeated drinking. This alcohol-produced depletion would aggravate the postulated preexisting serotonin deficit, setting up a "vicious cycle" in which the alcoholic repeatedly seeks to pharmacologically modify a central indoleamine deficit.

Figure 1. 5-HIAA in the CSF of type 2 alcoholic patients immediately after intoxication and during abstinence



NOTE: 5-HIAA = 5-Hydroxyindoleacetic acid; CSF = cerebrospinal fluid; ng per ml = nanograms per milliliter
SOURCE: Reference 5.

Figure 2. Alcohol and central serotonin: a possible "vicious cycle"



SOURCE: Reference 5.

A year later, we found that depressed patients with a history of alcoholism in first or second degree relatives had significantly lower 5-HIAA levels than depressed patients without family histories of alcoholism (*6*) (fig. 3).

Among other groups of sailors studied at the Bethesda Naval Hospital, those who had a history of impulsive-aggressive behavior problems also had low levels of 5-HIAA. Swedish investigators had earlier observed that depressed patients who attempted suicide were similarly deficient in the metabolite. Over the past decade, an odyssey of studies in Sweden, Finland, and the United States has added impulsive murderers and other impulsive, violent criminals, borderline personalities, arsonists, and violent suicides to the low 5-HIAA list. A profile has emerged of an antisocial personality characterized by low harm avoidance, high novelty seeking and low reward dependence—prone to alcohol abuse and linked to low brain serotonin activity.

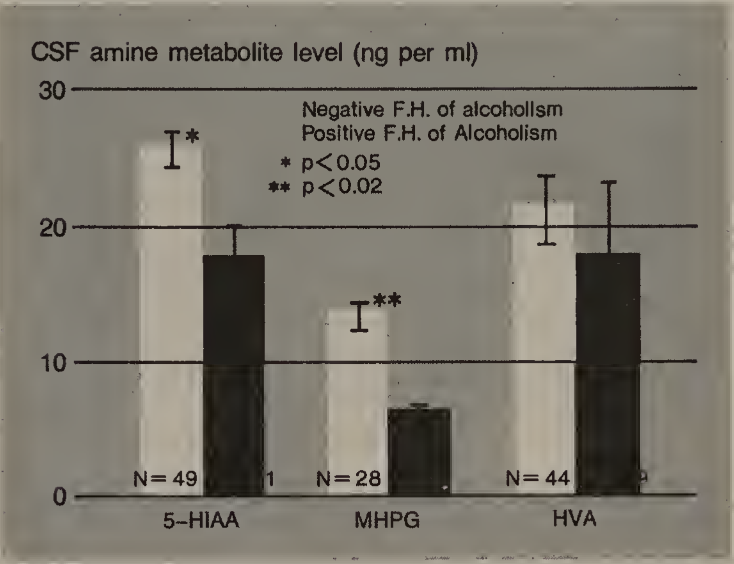
Studies in animals provide added support. Drugs or diets that deplete brain serotonin increase shock-induced fighting and mouse-killing behavior in rats. This effect can, in turn, be attenuated by drugs that inhibit reuptake of the transmitter from the synapse or otherwise enhance serotonin function.

All of this converging evidence dovetails with the genetically based subtypes of alcoholism characterized by C. Robert Cloninger of Washington University in adoption studies (*7*). In a recent review (*8*) of serotonin links to impulsivity, violence, and alcoholism, NIAAA investigator Alec Roy and colleagues built a convincing case for a serotonin-type II alcoholism connection. Type II alcoholism affects sons of alcoholics, requires no environmental triggers for expression of the gene, and is characterized by early onset in young adulthood, antisocial personality traits, craving for alcohol as a euphoriant, episodic binges, brawls, and arrests. Type 1 alcoholism—the more common form—affects both sexes, typically in mid-life, is linked to life stresses, anxious personality traits, and is marked by rapid development of tolerance, gradually increasing dependence, and less clear-cut genetic loading, at least in males. Mechanistically, this latter type probably relates more to the benzodiazepine/GABA receptor (gamma-aminobutyric acid receptor) complex. Thus, at least two discrete illness processes may be involved in what we call alcoholism.

Persons who fit the type II personality constellation have a tendency to develop low blood sugar during glucose tolerance testing and are also prone to disturbances of the sleep-wake cycle. Animal studies have shown that both glucose metabolism and circadian rhythm functions are regulated by the suprachiasmatic nucleus of the hypothalamus, which turns out to be densely innervated by serotonin neurons.

Perhaps it is more than a coincidence that Seasonal Affective Disorder (SAD) patients—who suffer disordered circadian rhythms as well as intense carbohydrate cravings with impulsive eating and drinking in winter—tend to have family histories of alcoholism (*9*). Indeed, my wife has observed a seasonal pattern of alcoholism in her work at Georgetown University, where they have had some experience treating the winter intensification

Figure 3. CSF monoamine metabolites in depressive subgroups



NOTE: CSF = cerebrospinal fluid; FH = family history; ng per ml = nanograms per milliliter; 5–HIAA = 5–Hydroxyindoleacetic acid; MHPG = 3–methoxy-4-hydroxyphenylglycol; HVA = homovanillic acid.
SOURCE: Reference *6*.

of alcohol abuse with the same high intensity, full spectrum lights used to treat winter depression. Among all neurotransmitters studied, serotonin deficits appear most likely to be involved in the pathophysiology of SAD, providing further rationale for the possible efficacy of light treatment in other serotonin-related disorders, such as alcoholism.

New research tools are now available to reexamine the serotonin hypothesis. These include specific pharmacological probes or challenge drugs, such as the serotonin receptor agonist MCPP (m-chlorophenylpiperazine) and the selective serotonin reuptake blocker fluoxetine. Preliminary studies suggest that such serotonin-stimulating substances can reduce alcohol consumption and also decrease the cognitive and memory deficits associated with alcohol. Another challenge strategy might involve administering a serotonin precursor substance such as tryptophan and examining effects on cortisol output as a way of gauging the integrity of neurohormone systems regulated by serotonin.

Kindling a Worsening Course

Another decade-old NIMH hypothesis about alcoholism now gaining currency proposes that the characteristic downhill course of the illness is "kindled" by repeated withdrawal episodes—a course that is analogous to that of electrically kindled seizures (*10*). Symptoms of tremor, shakes, seizures and delirium tremens become increasingly worse with each withdrawal experience, presumably because of a progressive deterioration in neuronal responses.

There is now evidence that repeated withdrawals may eventually trigger panic attacks and other anxiety disorders. The results of one recent study suggest that repeated withdrawals may eventually result in a hyperreactive noradrenalin system (*11*). It found that with successive withdrawals, the responsibity of noradrenalin (alpha 2) autoreceptors became blunted. Some of the investigators in this group are proposing a longitudinal, multicenter study "to determine the clinical significance of withdrawal-induced kindling and other central nervous system damage" (*12*).

Blocking Alcohol's Reinforcing Effects

To deal with highly reïnforced drinking behavior in vulnerable persons, we need to understand the cellular mechanisms for the initial effects of alcohol. As I noted earlier, Dr. Steven Paul and colleagues at NIMH have been studying the intimate workings of the brain's benzodiazepine system. A recent outgrowth of this work has been the discovery of a way to selectively block the intoxicating, reinforcing effects of alcohol in rats (*13*). The investigators have demonstrated that alcohol acts on the GABA-benzodiazepine-barbiturate receptor complex much like the benzodiazepines, stimulating uptake of chloride into the neuron. By injecting an "inverse agonist" which binds to and occupies the receptor (but which is without pharmacological effects of its own), they were able to block alcohol's ability to stimulate this chloride uptake and its attendant behavioral effects. Thus, they prevented that aspect of alcohol's effects which probably contribute most to repeating the behavior—the initial, mildly intoxicated calming effect.

The findings help to explain the anti-anxiety, sedative-hypnotic, and anesthetic properties of alcohol and may provide valuable insights into the mechanisms of alcohol tolerance and dependence. They also may lead to the development of medications that could be used to treat alcoholism in much the same way that naloxone and naltrexone are used in the treatment of opiate addiction.

Alcohol and Depression

As I noted earlier, in my clinical work with affective disorder patients, I have become increasingly impressed with the profound negative effect alcohol can have on the course of an affective disorder and with the profound interference the

' . . . it seems especially important to educate professionals who work with affective illness about alcohol abuse. By the same token, people in the alcohol field need to educate their colleagues about the affective disorders.'

drug can present to effective pharmacological management of patients. I feel we need to do a lot more to educate physicians about alcoholism and alcohol abuse. From my own experience, it seems especially important to educate professionals who work with affective illness about alcohol abuse. By the same token, people in the alcohol field need to educate their colleagues about the affective disorders. This is important because, although the two disorders overlap considerably, treatment approaches are quite specialized. There is now an array of effective treatments for depression. Issues of comorbidity with affective illness are still underappreciated, because the treatment systems for alcoholism and depression have tended to operate so independently of one another.

Borrowing new treatments from the other field may also prove fruitful. For instance, the previous discussion of alcohol and kindling may prove to be more than academic. We know there are many manic depressive patients for whom the anticonvulsant drug carbamazepine seems to be effective because of its antikindling properties. Perhaps anticonvulsants might be useful as an adjunctive treatment for certain patterns of episodic drinking.

Again, these remarks are simply some initial thoughts, examples of new alcoholism research opportunities offered us by the neuroscience revolution, drawn from my own research and clinical background. As ADAMHA Administrator, I expect to learn about the full range of biological and behavioral research in this exciting field. Indeed, the alcohol treatment and research communities are themselves showing an increased interest in each other's work—an encouraging development that will certainly benefit persons with this disorder.

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A Perspective on Science and Public Health Policy

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HISTORICALLY, ATTEMPTS to prevent alcohol-related problems have resulted in the formulation of public policies aimed at controlling the way in which alcohol is sold and used. Perhaps the most well-known example of this in the United States was Prohibition. Others have included State and local statutes governing public drunkenness, the sale of alcohol to minors, driving under the influence of alcohol, and a host of other measures designed to protect the public from the consequences of alcohol use.

Until fairly recently, alcohol-related public policies were based almost entirely on the ability of individuals and groups to convince Federal, State, or local governments of the need for such policies—no small undertaking, considering the stigma that has been attached to alcoholism and alcoholics. Evidence used to support claims of the impact and efficacy of public policies was based primarily on the experiences of recovering alcoholics, observations of alcohol treatment personnel, and common sense! (This path to the enactment of laws and policies to protect the public good is no different for alcohol than it has been for other public health areas or, for that matter, any area judged by society to be a public risk.)

In 1970, however, another factor entered into the alcohol-related policy equation—scientific research. Until that time, almost no research had been done on the causes and consequences of alcohol use. The creation of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) that year led to the beginning development of a national alcohol research program.

Over the next decade, as the alcohol field grew and matured, individuals, alcohol-related groups, and organizations with a variety of viewpoints began to look to science to support or refute various proposed and existing public policy measures. With this quest for scientific evidence came questions about the appropriate relationship between alcohol research and the formulation of public policies to prevent, reduce, or otherwise control alcohol abuse and alcoholism and, by extension, questions about the role of the NIAAA in formulating public policy.

NIAAA Activities

It is not possible to isolate the role of science in developing public policy about alcohol without an understanding of NIAAA's history. When NIAAA was established, the Institute had responsibility for all of the Federal Government's efforts to prevent and treat alcohol abuse and alcoholism. This included development of alcohol-related research, training of alcohol researchers and clinicians, and prevention and treatment programs.

By the end of the 1970s the NIAAA mission was changing from having direct responsibility for alcoholism treatment and prevention services, through support to State and local programs, to being a national resource for alcohol-related scientific, technical, and general information. The change from being the Federal focal point on all alcohol-related health issues to a national research institute was further accelerated by legislative changes in 1981 and 1986. The Omnibus Budget Reconciliation Act of 1981 transferred all responsibility for treatment services from the Institute to the States via an Alcohol and Drug Abuse and Mental Health Services Block Grant. In 1986, the Anti-Drug Abuse Act consolidated responsibility for alcohol and other drug abuse prevention and education services to a newly created Office for Substance Abuse Prevention in the Alcohol, Drug Abuse, and Mental Health Administration. Thus, for most of its history, NIAAA was responsible not only for a range of programmatic efforts related to alcohol abuse and alcoholism but also for developing national policies affecting the health aspects of alcohol use and abuse.

The present NIAAA mission is clear—to build the necessary scientific base to develop and improve methods to prevent and treat alcohol-related problems. As a research institute, the Institute

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supports and conducts basic, clinical, prevention, and epidemiologic research and supports research training programs for new scientists who wish to specialize in alcohol research. As a part of its research mission, NIAAA also conducts policy studies that have broad implications for prevention and treatment. A third major Institute function, to disseminate research findings, is accomplished by NIAAA through a number of mechanisms. These include the Alcohol Epidemiologic Data System; Alcohol Health and Research World, a professional journal distributed quarterly; Alcohol Alert, a publication designed to disseminate research findings quickly to alcohol clinicians; and specialized monographs and reports.

Role of Science in Policy Development

What is the nature of science? Science looks for answers. Science must be reliable and unbiased; scientists have a duty to report contradictory or negative evidence as well as confirmatory evidence. All scientific conclusions are tentative and can be modified or contradicted by further study. And science clearly has an important role in shaping public health policy decisions. Nevertheless, it is essential to understand that science and scientific evidence are rarely the *only* bases for public policy decisions.

In most situations, the public perception of a problem is a far more persuasive force in shaping public policies than is scientific evidence. For example, although scientific evidence of the harmful effects of cigarette smoking on health has been accumulating for more than 20 years, not until recently has the public perception of smoking as a major health hazard resulted in policies governing smoking in restaurants, office buildings, and other public places. The other determinants of public policies that play as great a role, if not greater than, scientific evidence are cultural, religious, and political values as well as economic interests. Consider the sheer complexity of developing policies dealing with a drug that is widely used, a drug that is legal for persons over the age of 21, a drug that many use without harm, and a drug that affects the national economy on both debit and credit sides of the balance sheet. These circumstances generate a complex mix of economic, religious, and political pressures that help shape public policy. For instance, there is strong, well-publicized scientific information on the genetic heritability of a predisposition to alcoholism. Yet, alcoholics continue to be labeled as morally weak—a view that led to a public policy decision by the Veterans Administration which defined alcoholism as ''willful misconduct.'' The scientific evidence of alcohol's detrimental effect on health, society, and the economy is ample; yet until the public perceives that alcohol abuse and alcoholism are public problems, public health policies will not be adopted to prevent or treat them.

It is important to understand that, as a science-dedicated institution, NIAAA does not *enact* social policy. We are a research organization that attempts to provide the data upon which social policies—some of which are politically sensitive—can be based. We can and do provide scientific information to individuals, groups, and others who may be advocates for one policy position or another; we cannot provide *scientific* justification for a particular position unless the research base is, indeed, supportive.

We do have research evidence related to some of the more sensitive social policy issues such as taxation, advertising, and college-age drinking. We know, for example, that alcohol consumption responds to price which, of course, raises the issue of taxation. Economic models have shown that if real prices were to change significantly for beer, for example—which they have not done for 30 years—several thousand young lives would not have been lost on our highways. Does this mean that the price will rise tomorrow? Of course not! Nor is it NIAAA's job to see that it happens. But the research that we have done in econometrics and on social issues provides evidence on which people can base their thinking about such issues.

Another sensitive public policy area is the advertising of alcoholic beverages. There is a widespread impression that we are smothered with advertising for alcoholic beverages on radio and television, in magazines and other print media, and on billboards and other advertisements in our communities.

Coupled with this impression is the belief that advertising has an effect on alcohol abuse and alcoholism. Yet the little research that has been done in the area—most of it in laboratory settings—does not demonstrate overwhelmingly that alcohol advertising has much effect. Because of the lack of scientific information on this issue, those in the alcohol field who want to change television and other forms of alcohol beverage advertising cannot really look to NIAAA science. They can, however, look to their own observations and to their political leaders—other potent elements in the social policy mix.

The social policy debate about college-age drinking creates a dilemma that might be resolved by science. For example, does restricting alcohol use on campuses cause students to go to a neighboring town to drink? If so, does this increase their risk for death and injury from an alcohol-related traffic accident? These are, of course, researchable topics that are appropriate for NIAAA research support.

Research also can lead to new ideas on preventing and treating alcohol-related problems. For example, there is a host of ideas on how to prevent children and teenagers from using alcohol. We already know that children in families with an alcoholic parent are at high risk for developing alcohol-related problems. Scientists are seeking to understand how we can identify these children by a variety of markers, such as changes in brainwaves, and other biological studies that may offer a clue to prevention and early intervention programs for high-risk youngsters. We also have evidence from psychosocial research that even at the early ages of 5 and 6, children who are particularly shy or aggressive in kindergarten are the ones most likely to get into trouble with alcohol and other drugs 10 years later. Using these scientific findings could well help school administrators and others who educate young children to design early age alcohol prevention programs.

Unfortunately, the evidence for some very popular kinds of approaches to preventing alcohol abuse and alcoholism is meager. For example, although enormous amounts of money and energy are going into school-based alcohol and other drug abuse prevention programs, when their effectiveness has been examined by rigorous scientific methods, the findings have been mostly negative. The data on programs that teach peer resistance are a little more positive, but the evidence comes only from short-term followup studies. The lack of scientific evidence, thus far, however, is no argument for stopping prevention and education programs pending the findings of research, but rather provides a strong rationale for continuing to conduct research on prevention activities.

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Science does not have all the answers and, in all probability, will never have all the answers for the formulation of public policies. Even when scientific evidence is strong, other factors will always influence events. They have in the past, and they will probably continue to do so in the future. We are not strangers to policy decisions that may not be consistent with what science has found. Alcohol scientists have, can, and should communicate research findings to others in the alcohol field as a part of various policy debates; research findings are not useful if they are known only to researchers. Those who are involved in policy formation, however, should not expect science to support only a particular position or to be the singular determinant of a policy direction.

Each of us working in the alcohol field shares a common purpose. Whether scientist, clinician, policymaker, trainer, or volunteer, we all work to help reduce the suffering associated with alcohol abuse and the disease of alcoholism.

Theories and Models Supporting Prevention Approaches to Alcohol Problems Among Youth

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Synopsis

The Alcohol, Drug Abuse, and Mental Health Administration's Office for Substance Abuse Prevention (OSAP) was established to initiate programs to provide prevention and early intervention services for young people, especially high-risk youth. OSAP's starting point was the theories and models that provide the background body of knowledge. The models summarized here guide new prevention efforts and provide a framework for analyzing diverse experiences in the field. The goal has been to develop strategies based on theories and models of prevention that can reverse or prevent adolescent alcohol use.

Among the psychosocial models, research in social learning theory is the theoretical basis for prevention efforts using the team approach among individuals, small groups, families, and communities. A prevention technique based on cognitive dissonance theory proposes verbal inoculations to establish or strengthen beliefs and attitudes, helping a young person to resist drinking, which may be in conflict with another, more desirable goal. In the developmental concept adolescence is a period of role confusion out of which the person's identity should emerge. Prevention efforts built on this view seek to help adolescents to form positive identities by achievement as students, athletes, and in community roles. Behavioral intention theory provides a framework for understanding the role of perceived social norms in directing behaviors. In the social development model, prevention programs should create positive peer groups and ensure that the social environment does not give mixed messages. Health behavior theory is the basis for prevention strategies directed toward a person's entire behavior instead of one aspect. The stages of the drug involvement model form the basis for prevention programs providing early intervention directed at the so-called gateway drugs.

Among the communications models, the health promotion concept advocates a comprehensive approach in developing health campaigns and attention to the five major elements of the communications process. Mass media campaigns based on the communication-behavior change concept address the steps required to move a target population from initial awareness of interest in a problem to the adoption and maintenance of advocated attitudes or behaviors.

Among public policy models, researchers have concluded that higher real prices on alcohol and restricted availability have the effect of lowering alcoholic beverage consumption among young people and the incidence of heavy and frequent drinking. Raising the minimum age for purchase has been found to reduce the rate of automobile accidents involving young persons. Essential components of prevention strategies and approaches are presented.

IN 1983, the World Health Assembly resolved that problems related to alcohol consumption rank among the world's major public health concerns and constitute a serious hazard for human welfare (*1*). Dr. Otis R. Bowen, the Secretary of Health and Human Services, reiterated this concern last fall by issuing a number of initiatives that address this nation's alcohol problems.

In addition, over the years, the National Institute on Alcohol Abuse and Alcoholism, and the National Institute on Drug Abuse, have warned of a steady rise in alcohol and drug use by youth. As a result of this mounting concern, the Alcohol, Drug Abuse, and Mental Health Administration's (ADAMHA) Office for Substance Abuse Prevention (OSAP) was established to initiate programs to provide prevention and early intervention services for young people, especially high-risk youth. The models presented here form the background body of knowledge that has been OSAP's starting point.

During the last decade, there has been an emerging consensus that the public health approach can provide a useful conceptual framework for developing alcohol and drug abuse prevention strategies. The focus of this paper is the prevention of alcohol problems using a public approach, specifically theories and models that provide the knowledge base for prevention programs targeted toward youth. The models summarized guide new prevention efforts and provide a framework for analyzing diverse experiences in the field. The models permit assessment of prevention approaches and generalization from one set of circumstances to another.

Within the public health approach to prevention, there is room for multiple theories and combinations of theories. The public health approach originally derived from epidemiologic studies of communicable diseases, indicating that proper prevention planning requires knowledge of the host (or individual person), the agent (in this discussion, alcohol), and the environment (the social milieu). Prevention, as we know it today, also requires knowledge of the interaction between the three factors.

Because prevention in public health is an evolutionary field that is continuously growing from the thinking and experiences of researchers, planners, practitioners, and evaluators, the current knowledge base will change, expand, and emerge in new combinations, providing better tools with which to address the prevention of alcohol problems.

The theories that seek to explain behavior and changes in behavior are taken largely from psychology. Until recently, most theoretical work in alcohol abuse prevention has been aimed at identifying the likely antecedents and correlates. More recently, the goal has been to develop strategies based on theories and models of prevention that can reverse or prevent adolescent alcohol use (*2*).

Psychosocial Models

Social learning. Albert Bandura's social learning theory is probably the most widely used among current prevention program planners. According to Bandura, learning is acquired and shaped by positive and negative reinforcements (rewards and punishments), as well as by observation of other people's behavior (*3,4*). Thus, people can anticipate the consequences and shape their own behavior to earn rewards and punishments.

Bandura recognized the potential for using modeling as a way of directing and changing behavior. We observe the behavior of others in person, on television, and in movies, and we become more likely to adopt that model's behavior based on the attractiveness of the model (*3, 4*). Bandura's early work indicated that children readily imitate aggressive models as well as more positive models and that status envy was an important factor (*5, 6*).

Later, his theory formed the underpinning for the use of "near peers" to transmit messages to somewhat younger children by depicting behavior and attitudes for young people to imitate, such as saying "no" to alcohol (refusal skills). In addition to the use of "near peers" in television mass media efforts, Bandura's work most likely is the theoretical basis for prevention efforts using the team or buddy approach, teaming individuals, small groups, families, and even communities, in which new health related behaviors can be modeled and reinforced, helping to set new norms.

Cognitive dissonance. Much of William McGuire's work in psychology is based on Festinger's concept of cognitive dissonance (*7*), which describes a tendency of humans to harmonize expectations about people and experiences with them. In other words, we want our beliefs to be in harmony with our experiences.

Cognitive dissonance theory holds that people want their personal attitudes and beliefs to be compatible with their own behavior (*7*). If they are not, there is "cognitive dissonance" that a person will want to eliminate.

As a prevention technique, McGuire proposed that certain "pretreatments" would establish or strengthen beliefs and attitudes with which a person's behavior would have to harmonize to avoid cognitive dissonance (*8*). "Cognitive inoculation" is one of these pretreatments. He found that verbal "inoculations" had certain immunizing effects against strong counter-arguments, and strengthened the subjects' ability to defend their beliefs (*9*).

When applied to the use of alcohol and other drugs, cognitive inoculation aligns a person's beliefs and behavior with regard to these substances. For instance, if a teenager believes that drinking will diminish athletic ability, and places a high value on athletic ability, resolution of dissonance would require that the teenager either abstain from drinking or place a lower value on athletic ability (*4,10*).

Another pretreatment approach requires that a behavior commitment be made; the commitment often is in the form of a contract or public announcement of one's beliefs and intentions ("I will not use alcohol until I am of age"). McGuire found that a commitment made to others was stronger than a private commitment (*2,10*).

Social inoculation. Richard Evan's social inoculation theory extends McGuire's, and addresses social influences, beliefs, and attitudes that create pressure on a young person to use alcohol and other drugs (*1,11*). Such pressures include peer pressure or joining a group that exerts pressure on new, younger group members.

Programs based on social inoculation theory teach students to resist social pressures. The popular "Just Say No" program is based on social inoculation theory (*2*). Students first are taught about the pressures that they can expect from peers to use alcohol and other drugs and then they are taught various ways to get out of uncomfortable peer pressure situations. In addition to instructing students to say "no," the "Just Say No" groups teach alternative responses, such as "I'm not interested." Many programs based on the social inoculation theory use modeling, as suggested by Bandura, to teach peer resistance skills. Others encourage public commitments from students as an added incentive to resist negative peer pressure. Social reinforcement often includes the entire group (*4*).

Although results often are mixed in prevention evaluation, one point seems clear. Peer pressure is one of the most significant factors in alcohol use and abuse, and successful prevention strategies will address this fact. For instance, the most important correlate of beer drinking is peer influence, followed by exposure to advertisements for beer (*12*). Peer influence has been found to have a significant impact on consumption levels and alcohol use behavior (*13*).

Developmental concept. Eric Erickson defined the implications of years of clinical observations in the landmark book "Childhood and Society" (*14*), in which he argued that psychological development continues throughout life and that each stage has a positive and negative component. In addition, each stage has a "task" or psychosocial "crisis" associated with it that must be successfully resolved.

For instance, in the first year of life, a child must develop a sense of trust rather than mistrust of the world around him or her. A child who is fondled and cuddled develops a sense of the world as a safe place to be. If the basic trust as opposed to mistrust issue is not resolved during the first year of life, it arises again at each successive stage of development. The core of Erickson's theory is that each developmental crisis must be resolved at each successive stage of development. At the least, if these tasks are not mastered at the appropriate stage of development, the individual will be at a disadvantage in making subsequent adjustments (*15*).

Erickson said that the major crisis confronting an adolescent is associated with establishing his or her identity and avoiding identity diffusion. He believes that the interpersonal dimension that emerges during this period has to do with a sense of ego identity at the positive end and a sense of role confusion at the negative end. In other words, adolescence is a period of role confusion out of which the person's identity should emerge. Certain behaviors, such as being sexually active, smoking, and drinking alcohol, are considered deviant because they are essentially adult behaviors which are being exhibited at an early stage of the life cycle (*3*).

Because the adolescent's body is changing rapidly, the developing self-image coincides with the identity crisis. As adolescents break away from the close guidance of their parents, they seek support elsewhere, usually with a peer group. In the developmental stage, an adolescent may view smoking, or the use of alcohol and other drugs, as a way of expressing a growing sense of independence.

Prevention programs that seek to build upon the developmental theory ought to assist adolescents in forming positive identities as athletes, students, community or hospital volunteers, environmentalists, or peer leaders of younger students. The adolescent, in Erickson's view, is "an impatient idealist" who believes that it is as easy to realize an ideal as it is to imagine it. A prevention strategy could capitalize on this theory by helping adolescents to form positive identities around their ideals.

Additionally, prevention strategies should be developmentally appropriate for the targeted age group. An example is reported by Funkhouser (*16*) in testing materials for 8- to 12-year-olds who do not yet think in abstract terms. Slogans encompassing the phrase "alcohol-free" meant, to them, "free alcohol." Successful prevention strategies are those which are appropriate for the target audience's stage of development.

Behavioral intention. Martin Fishbein and Icek Ajzen developed a system for measuring attitudes as well as subjective norms (*17*). Their behavioral intention theory postulates that, when properly measured, a person's attitudes and subjective norms about a behavior can be used as predictors of behavior.

Attitudes are the beliefs a person holds about the outcome of a behavior, along with the value he or she places on that outcome (*4*). The most prominent dimension of attitude is like-dislike, the tendency to accept or to reject or to seek to avoid (*18*). Subjective norms are the person's assumptions about the views of significant others regarding the behavior, along with the person's motivation to comply with these views.

For attitudes and subjective norms to be valid predictors of behavior, they must be measured in terms of specific behavioral situations. For example, a person drinks (action) beer (target) in a car (context) on a weekend (time). Attitudes and subjective norms must be assessed to keep the action, target, context, and time in focus (*4*).

The theory provides a good framework for understanding the important role that perceived social norms play in directing behaviors. For example, adolescents generally perceive a prevalence of alcohol and drug use among their peers that far exceeds actual consumption (*19*). Students who overestimate the use of alcohol and other drugs by their peers may view that use as "normal" and become more accepting of drinking and other drug use. Changing behavior, therefore, means not only changing norms but the perception of norms. Correcting overestimates can be a useful prevention message (*20*).

Another indication that the theory provides a good framework for understanding the important role that perceived social norms play in directing behaviors came from a recent study by Lloyd Johnston and coworkers (*21*). Although this study dealt with marijuana use, the main finding may be important in suggesting a direction for future alcohol use prevention efforts. The researchers found that the decline in marijuana use since 1979 among high school seniors is most likely because of a change in norms and their perception of norms.

To be precise, negative attitudes toward marijuana use among high school seniors are increasing. Disapproval by peers and an increase in the perception of risk involved in using marijuana are most likely the deciding factors that have led to decreased marijuana use (*21*). According to Bachman and coworkers, increasing proportions of high school seniors perceive that their friends disapprove of marijuana use, and increasingly, information concerning harmful consequences is believed. These "changes in the social environment" suggest that future prevention efforts may effectively emphasize perception of harm and social disapproval.

Social development model. David Hawkins and coworkers developed a model that seeks to address key risk factors for alcohol and other drug use at developmentally appropriate points (*22*). According to the model, the development of bonds of attachment between adolescents and social units, such as families, schools, and groups of friends, creates a system of rewards and punishments. When the social unit's expectations are clear and consistent, young people's behaviors will follow the norms because they feel socially bonded to the units (*4*).

According to the social development model, prevention programs should create positive peer groups and ensure that the social environment does not give mixed messages. An example of this type of program is the Canadian Four Worlds Development Project, created by Phil Lane and tribal elders for Native American communities (*23*). The project promotes the development of core groups within Native American communities, groups that will "establish new structural patterns around which the life of the community can organize itself—patterns that are more life-preserving, life-enhancing and development-promoting than those currently being utilized."

Centered in Alberta, the project's founders believe that the "core of human development process is the people who, by their unity of vision, their personal commitment to growth, and their leadership (by example), form the pattern which other people cluster as positive changes take place in the community."

Health behavior. The health behavior theory is based on Karl Lewin's field theory and emphasizes the uniqueness of the individual as well as the properties of the group (*4*). Perry and Jessor described the variables that influence behavior in terms of spheres and propose that health is comprised of four domains; physical, psychological, social, and personal. Since a single behavior can effect several health domains, the relationships among them must be examined fully in terms of prevention strategies (*24*).

Because adolescents tend to engage in several risky health-compromising behaviors at the same time rather than one, Perry and Jessor recommended that prevention strategies be directed toward a person's entire behavior repertoire (*24*). The promotion of health-enhancing behaviors through stress reduction, elimination of smoking tobacco, and regular physical exercise can be viewed as complementary strategies in a field theory-based health promotion program.

Stages of drug involvement. Denise Kandel and coworkers described alcohol and other drug use as a process of clear-cut stages largely determined by a matrix of social relationships (*25,26*). Because of the longitudinal nature of their studies, they could determine that different influences are involved at different stages, with situational and interpersonal factors being most important for initiation into alcohol and other drug use behavior. Psychological factors were found to be most important for increased involvement or participation in that behavior.

Three specific stages of drug use described by the researchers are use of alcohol, use of marijuana, and use of other illicit drugs. Distinct predictors mark initiation into each stage of use. The most important predictor of alcohol use is involvement in minor delinquent activities. The researchers documented the role of alcohol and other drugs as a gateway to increased risk of progression to greater or more frequent use (*27*). The use of alcohol and illicit drugs at a young age predicted an unstable work history, as observed during the 9-year study period (*27*).

An example of prevention programs based on Kandel's work is those attacking gateway drugs. OSAP supports prevention efforts that have special interventions at early ages for youths who smoke cigarettes or use chewing tobacco, alcohol, or marijuana. OASP also supports special interventions at an early age for youths who have favorable attitudes toward smoking, drinking, or drug-taking subgroups.

Drug use as a deviant response. One popular model explains drug use during adolescence as deviant behavior. Kaplan and coworkers (*28*) proposed that such deviant responses often are triggered by an adolescent's membership in groups which have deviant response patterns that act effectively to boost self-esteem. How this model could be useful in explaining alcohol use is still unclear; however, it is interesting because it involves both the individual's self-rejecting attitude and membership in a negative reference group.

Sensation seeking. Zuckerman's research explored the relationship between an individual's need for sensory stimulation and a host of behaviors, among them the use of alcohol and other drugs (*4,29*). He observed that all individuals seek varied experiences and stimulation but have different optimal levels of arousal. Using alcohol or other drugs, eating, smoking cigarettes, and engaging in sexual activity are examples of sources of sensory stimulation. The primary functions of sensation seeking are to provide new experiences, reduce boredom, facilitate disinhibition, and offer adventure.

The most important demographic correlates with sensation seeking are age and sex. Thrill or adventure seeking declines after age 30, and males generally score higher than females on sensation seeking. Young males' higher rates of drinking and driving could perhaps be explained by this model.

However, to believe that prevention is simply providing opportunities for recreation would be simplistic. An example of a misguided prevention program was provided by Milam and Ketcham (*30*). A large grant for Eskimo villagers to construct recreation centers was based on an assumption that the people were bored and would stop drinking if provided with a place for recreation. But the alcoholism rates were already high in the villages and, as the researchers noted, "boredom is not a cause of alcoholism"; the villagers primarily needed treatment centers. OSAP recommends that intervention and treatment programs be established in addition to prevention efforts across the country; one is not a substitute for the other.

Communications Models

Health promotion. Bettinghaus expanded the original knowledge-attitude-behavior continuum by using measures of behavioral intention rather than generalized attitudes (*4*). In addition, he advocated the use of a comprehensive approach in developing health campaigns. Key elements that should be addressed include countermeasures (antismoking efforts that compete with prosmoking print media messages); difficulty (losing 50 pounds is more difficult than reducing salt intake by 10 percent); addictive properties (tobacco, alcohol, and other drugs); and social pressures (from peers).

Finally, he proposed the use of McGuire's information processing model, which argues that moving between the elements of the knowledge-attitude-behavior continuum demands that processing time on the part of individuals, as well as attention to a set of elements within a communication matrix. The matrix includes, at a minimum, paying attention to the five major elements of the communications process, which are source, message, channel, receiver, and destination (*4*). The model has been used by OSAP in planning media campaigns for particular target audiences. OSAP prevention efforts include the use of mass media to support and reinforce community efforts.

Communication and behavior change. Maccoby and coworkers studied mechanisms used by mass media campaigns to change behavior and defined the steps required to move a target population from initial awareness of interest in a problem to the adoption and maintenance of advocated attitudes or behaviors (*4,31*).

The first step involves attracting attention and focusing it on specific issues and problems, a process referred to by mass media researchers as "agenda setting," which, for major national campaigns, has been accomplished by broadcast media. Once a subject is on the public agenda and perceived as an important issue, a campaign must explain the issue in a way that is personally relevant to individuals in the target audience. The target population also must be given some positive incentives or encouragement to change their behavior, and taught how to modify risk-related behaviors ("training"). Solomon pointed out that it is important for the person to understand the price of engaging in risk reduction behaviors from an individual point of view (*32*).

Finally, if newly acquired habits are to be maintained, self-cuing at appropriate times and places is critical. Prevention strategies are designed to take into account the steps in the process, as well as the potential influences operating at the personal, community, and economic levels (*4*).

In addition, Solomon advocated extensive use of audience segmentation and the development of multiple products that are targeted to each primary and secondary audience (*32*). OSAP has applied this principle in its "Be Smart! Don't Start!—Just Say No!" campaign.

Persuasion-communications. McGuire has worked extensively on the theory underlying communication campaigns (*33,34*). He believes that the more successful campaigns are those which focus on the availability of solutions rather than on the seriousness of the problem (*33*). He found that such techniques as defining specific audiences and targeting messages may increase the effectiveness of public communications campaigns, but the techniques have limited efficacy for changing the audience's health-related behavior (*35*).

McGuire described components necessary to construct a communication capable of changing attitudes and behavior, along with successive responses people must make if they are to "yield" to the communication (*4*).

According to McGuire, the effectiveness of a communication campaign depends on its ability to lead an audience through a 12-step process. The process starts with exposure to the communication and proceeds through learning how to incorporate the target behavior in one's life, and being able to make decisions based on the retrieval of the information. The process concludes with reorganizing one's related beliefs and "engaging in postbehavioral activities" or consolidation (*4,33*).

Public Policy Models

Price policies. Growing evidence indicates that the price of alcohol influences both alcohol consumption and alcohol-related problems (*35*). Cook (*36*) and Cook and Tauchen (*37*) found that relatively small increases in the price of distilled spirits (increases in State taxes) reduced not only consumption of distilled spirits but also death rates from liver cirrhosis and automobile crashes.

More recently, Coate and Grossman (*38*) concluded that higher prices for beer, the most popular alcoholic beverage among youths, would reduce not only the numbers of young persons who drink but also the incidence of both heavy and frequent drinking. A recent longitudinal study that compared various regulatory and availability restrictions (such as hours of sale) concluded that raising the prices for alcohol had the greatest effect in lowering consumption (*39*). Changes in price policies appear to be a very effective means of reducing alcohol consumption.

Minimum drinking age. Research indicates that lowering the minimum age for alcohol purchase is associated with an increased rate of automobile crashes among young people, and that raising the minimum age reduces that rate (*4,35*). Researchers studied the automobile fatalities of 26 States that raised the minimum purchase age between 1975 and

'In the developmental stage, an adolescent may view smoking, or the use of alcohol and other drugs, as a way of expressing a growing sense of independence.'

1984. The findings showed a 13 percent reduction in night-time fatal crashes among 18- and 19-year-old drivers, the most affected age group. The effect was consistent across regions and stable over time (*40*).

Research into the physical availability of alcoholic beverages continues to support the hypothesis that a reduction in availability through various means, including age restrictions, hours and days of sale, and raising the minimum age, is correlated with lower rates of use and a lower incidence of related problems. Changes in these environmental factors, therefore, appear to be a very effective means of reducing alcohol consumption and problems. OSAP is making the research results available for implementation by those developing prevention programs.

Conclusion

No single approach has been identified as effective for preventing use of alcohol by youth. In fact, different factors in separate programs appear to be effective in certain communities with certain age groups. A multifaceted approach to prevention is necessary.

Dr. Sheila Blume, an author and scientist in the alcohol field, said "If we ever develop a rational prevention policy, it will have hundreds of items in it . . . they will all have to be done" (*41*).

For maximum effectiveness, a prevention strategy needs to address parental and peer influences, teachers, and community leaders; norms; marketing and availability of alcoholic beverages; and alcohol-related laws, regulations, and policies (*19*).

OSAP was established within ADAMHA by the Anti-Drug Abuse Act of 1986 to develop a multidimensional prevention program with a fresh perspective and new resources. OSAP activities are based on the knowledge that prevention can be only as effective as the efforts undertaken at the State and community levels. Therefore, OSAP activities are focused on providing national leadership to strengthen and support local efforts.

Briefly, the OSAP mission is to (*a*) review government policy related to alcohol and other drug abuse, (*b*) operate a clearinghouse, (*c*) operate a grant program, (*d*) support the development of model programs, (*e*) conduct prevention workshops, (*f*) coordinate research findings, and (*g*) develop prevention materials.

Prevention planners should focus on the multiple factors that contribute to alcohol and other drug use by youth. Community leaders should take into consideration existing community efforts and in their programs address ways in which various programs can interrelate. OSAP recommends a systems approach to prevention, viewing the community and the environment as interconnected parts, each affected by the others. The goal of any community in prevention is to make the parts work together (*20*).

Although more research is needed to determine which strategies and approaches work best, the following (*19*) are essential components:

- Communicating a clear, nonuse message for youth through all community channels, policies, and practices
- Role modeling of moderate, low-risk use of alcohol by adults of legal age
- Skill building to enhance social and interpersonal communication skills, peer resistance problem-solving, media promotion efforts and portrayal analysis, and ability to ask for help
- Promoting bonding and attachments to family, peers, school, and religion, and belief in general social norms, values, and expectations
- Increasing the perceived benefits of health-enhancing behaviors and decreasing the perceived benefits of health-compromising behaviors
- Providing referral, counseling, or treatment services for children or families in need of help.

Alcohol is the most widely used drug among American youth. Preventing its use by young people requires an integrated, comprehensive, systems approach that involves the entire community—parents, older youth, educators, health and social service professionals, religious organizations, government agencies, recreation and service groups, and businesses. Strategies must be reinforced through the mass media and through one-on-one peer programs to enable behavior change and maintenance to occur. If we apply these strategies consistently over time, we could see a new generation of drug- and alcohol-free youth.

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Screening for Alcoholism: Techniques and Issues

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Synopsis

Alcoholism is an often overlooked health problem because alcoholics usually do not seek treatment for their drinking problems. They do, however, seek general medical care for other health reasons, and a number of screening techniques have proven useful for identifying alcoholics. The advantages and disadvantages of self-report, as well as biochemical techniques that have been found effective in screening for alcoholism, are discussed.

We recommend that future research be aimed at developing quick, accurate, and inexpensive screening devices that also can evaluate the severity of the alcohol problem. Ideally, screening procedures would discourage feigned responses, differentiate between drinking and consequences of drinking, and permit the identification of subtypes of alcoholics. Better understanding of the types of errors made by common screening instruments would enable researchers to construct an optimal sequencing strategy for screening for alcoholism.

ALCOHOLISM is one of our society's most overlooked health problems despite its significant medical, economic, social, and legal consequences. Only about 15 percent of the alcoholics in this county ever enter any type of treatment for alcoholism (*1*).

Although alcoholics rarely volunteer for treatment for alcoholism, they do seek and receive other kinds of care. For example, a study sponsored by the National Institute of Mental Health has shown that, within a 6-month period, almost 70 percent of individuals with alcohol problems make at least one ambulatory care visit. About 90 percent of these visits are to medical providers rather than to mental health specialists (*2*). An estimated 15 to 30 percent of medical and surgical patients in general hospitals satisfy clinical criteria for the diagnosis of alcoholism (*3*). Therefore, general medical care settings appear to be appropriate sites for screening for alcoholism.

Two types of alcoholism screening instruments are currently available to health care practitioners. There are brief interviews, or paper and pencil tests, in which the patient gives a self-report of his or her drinking behavior and its consequences. There are laboratory measures in which enzyme levels and other biochemical test results are analyzed to determine if, individually or in combination, measurements deviate from expected values. Values outside the normal range frequently indicate alcohol-related pathology, especially liver damage, although such a determination may only be possible when other medical conditions and the use of certain medications have been ruled out.

We review examples of both types of screening procedures, discuss research and clinical practice issues involved in screening for alcoholism, and outline important research needs for alcoholism screening.

Measures of Alcoholism

Self-report measures. Four instruments frequently used to screen for alcoholism are the Michigan Alcoholism Screening Test (MAST), the Addiction Severity Index (ASI), the MacAndrew Scale, and a four-question screening instrument called CAGE. Another instrument, recently developed, is the only one intended for use with adolescents.

MAST is one of the oldest and most commonly used self-report measures of alcoholism (*4*). MAST consists of 25 items dealing with drinking patterns; social, occupational, and medical aspects of drinking; and previous attempts at treatment. Items are differentially weighted based on their degree of relationship with alcoholism. Hit rates in screening are generally expressed as sensitivity and specificity percentages. Sensitivity refers to the percentage of true positives, and specificity to the percentage of true negatives. Hit rates differ somewhat as a function of the population studied. Hedlund and Viewig, in their review of the literature on MAST, report values in the .80s and .90s (with four studies reaching 1.00) for specificity with a cut score of 5 (*5*).

Efforts to further improve upon MAST include a version, developed by the Veterans Administration, which also asks about the time at which various symptoms were experienced; several abbreviated forms of the inventory; and the Self-Administered Alcoholism Screening Test (SAAST) (*6*). SAAST differs from its parent by including an additional nine items on symptoms, internal checks for consistency of responses, and the use of simple unit weights for scoring items. SAAST has been reported to have a sensitivity of 95 percent and a specificity of 96 percent (*7*). SAAST appears to have excellent validity (*7*) even when administered to spouses who report on their alcoholic partners.

ASI differs from MAST in two key respects. ASI attempts to determine the severity of alcoholism, based on collateral associated problems. ASI seems to have applicability for matching patients to appropriate levels or intensities of rehabilitation services. ASI was developed by McLellan and coworkers to serve as a quick means of establishing a psychiatric or substance abuse diagnosis and as a research instrument to assess treatment-related changes in seven problem areas (alcohol, drugs, medical, employment and support, legal, family and social, and psychiatric) (*8*). Ratings are based on objective and subjective information provided by the client and cover two time periods, the past 30 days and the client's entire life.

ASI seems to have both good inter-rater reliability and test-retest reliability. The measure takes about 45 minutes, and can be used both as a screening instrument and as a means for estimating the severity of drug or alcohol abuse. In addition, it has been used to assist clinicians in determining the intensity of treatment needed. McLellan found that ASI differentially predicted success in inpatient or outpatient treatment as a function of the severity of the patient's adjustment difficulties (*9*).

CAGE is a self-report screening for alcoholism and the briefest validated screening interview available. CAGE consists of four questions: "Have you felt the need to *C*ut down?"; "Do you feel *A*nnoyed by people complaining about your drinking?"; "Do you ever feel *G*uilty about your drinking?"; and "Do you ever drink an *E*ye-opener in the morning to relieve the shakes?"

Despite CAGE's simplicity, its levels of sensitivity and specificity are quite acceptable (that is, at a cut-score of two affirmative responses, 75 percent of alcoholics are correctly identified as alcoholics and 96 percent of non-alcoholics are correctly identified) (*7*). CAGE appears to be particularly valuable in a busy medical setting where there is little time to interview patients.

The MacAndrew Scale is unique among self-report screening devices in that it does not ask directly about drinking or effects of drinking. This aspect is important because alcoholics may fail to recall amounts consumed or may fail to associate adverse life consequences with drinking. The scale was developed on purely actuarial grounds (*10*). Those items on the Minnesota Multiphasic Personality Inventory (MMPI) which were answered differently by male outpatient alcoholics than by male outpatient psychiatric patients were selected for inclusion in the scale. MacAndrew found that the two groups differed in their responses to 49 non-alcohol-related items. Using cut scores from 24 to 28, the MacAndrew Scale has yielded good discrimination between alcoholics and nonalcoholics in a wide range of subsequent studies. The scientific literature on the measure has been critically reviewed by Preng and Clopton (*11*). While the MacAndrew Scale is in general much less valid than the other pencil-and-paper measures, it appears to have some advantages in that it does not seem readily "fake-able" and may identify specific types

> *'Screening can identify persons who suffer from alcoholism, increasing the likelihood that they will receive effective treatment. While this goal may appear to be ambitious, screening can be part of minimal intervention treatment, which may in fact be quite effective, particularly with those who are in the early stages of alcohol abuse . . . '*

of alcoholics such as the hedonistic, extraverted, and nonconforming. Additionally, it can be derived as a by-product of a common psychological test without requiring additional testing effort.

"Drinking and You" is a new self-report instrument that was developed for use specifically with adolescents (*12*). It is mentioned here because most screening instruments for adolescents are simply modifications of instruments used for adults. Normative work on the measure is nearing completion. The test has already been validated against blind clinical judgments, and test-retest reliabilities have also been derived. Convincing convergent and discriminant validity indices have been obtained. "Drinking and You" is a 24-item instrument, appropriate for adolescents aged 12 to 17 years, and requires about 10 minutes to complete. The items tap four conceptual dimensions: loss of control of drinking, social symptoms, psychological symptoms, and physical symptoms.

Additional keys are available to distinguish self-medicating drinking and "aggressive-rebellious" drinking. Overall scores on the test allow adolescents to be classified at three levels of alcohol abuse severity from "minor or no problem" through "moderate problem" to "severe problem." In a recent study this instrument correctly identified 88 percent of adolescents with alcohol problems and 82 percent of those without problems when a dichotomous category distinction was used.

Biochemical measures. There are two basic approaches to identifying biochemical markers of alcoholism. The first relies on routinely requested clinical laboratory tests and the other on specialized laboratory tests.

Statistically significant relationships have been observed between reported alcohol consumption and routinely requested clinical laboratory tests (*13–16*). Several clinical tests have been described as useful in identifying alcoholics. These are mean corpuscular volume, or MCV (*17, 18*), and gamma-glutamyl transpeptidase, or GGT (*19, 20*). Such measures alone, however, have been shown to lack diagnostic specificity (*21*). This is not surprising in that it is rare that a medical condition can be defined by a single pathognomonic sign or symptom.

The use of multiple-test approaches has been demonstrated to have potential utility (*22–24*). Combinations of commonly requested clinical laboratory tests have been used to identify persons who have consumed alcoholic beverages in amounts that alter physiological processes (*25*), or that result in organ dysfunction (*26*), such as biopsy-verified liver disease (*22*). Correct classification ranges from 80 to 100 percent accuracy, depending on the population studied and the technique employed.

Some clinical investigators have advocated the use of specialized laboratory tests to diagnose alcoholism, such as 2,3-butanediol (*27*) and alpha amino-n-butyric acid (*28*), and several new ones have been proposed.

Two enzymes in blood platelet membranes, monoamine oxidase and adenylate cyclase, have been found to be altered in alcoholics, compared with controls matched for sex and age (*29*). *In vitro* inhibition of activity of platelet monoamine oxidase by 400 mM ethanol was more pronounced in alcoholics than in controls. It was also reported that *in vitro* stimulation of platelet adenylate cyclase by various agents was less in alcoholics. Analyses of these two enzymes resulted in correct classification of 75 percent of alcoholics and 73 percent of nonalcoholics.

Transferrin is a glycoprotein involved in iron transport. Excessive alcohol consumption has been associated with increasing levels of carbohydrate-deficient form of transferrin, or CDT (*30*). In several large clinical trials, evaluation of CDT has identified excessive alcohol consumption with 95 percent accuracy, suggesting that it may prove useful as a marker for alcohol abuse (*30, 31*). CDT has been demonstrated to be useful in identifying black and Puerto Rican alcoholics (81 percent sensitivity and 91 percent specificity). Unfortunately, this study also shows that CDT is often elevated in some nonalcoholic patients with primary biliary cirrhoses (*32*).

Acetaldehyde is the major metabolite of ethanol and has been shown to bind covalently to certain proteins forming immunogenic condensation products. Levels of some forms of these acetaldehyde adducts (AA) have been reported to be higher in sera of alcoholic patients (*33*). For example, hemoglobin has been shown to form a stable adduct with acetaldehyde *in vitro* (*34*) and has been used successfully to identify alcoholics (*35*). Moreover, recent data have demonstrated that a specific hepatic protein-AA is present in the liver during periods of ethanol consumption (*36*).

Issues

Although at least 400 studies have reported on screening for alcoholism, there still are important issues to be addressed.

Purpose of screening. One area of inconsistency in the screening literature relates to the nature and extent of screening as a construct. Some instruments are seen as screening instruments when their purpose goes beyond simple recognition of alcoholism to the assessment of its type and severity. Examples include the Alcohol Use Inventory, the Alcohol Dependence Scale, and the Severity of Alcohol Dependence Questionnaire (*37*). A distinction can be made between screening (identification of a possible problem in a large unselected group of persons), case-finding (more precise specification of a problem among persons who are known to suffer from some kind of difficulty), and diagnosis (determination of the nature and severity of a problem with recommendations for treatment). The practitioner concerned with alcoholism in patients must decide the particular goal of screening. This decision is related mainly to whether or not the patient will be referred to a specialized agency (and the capabilities of the agency) or treated in-house.

Value of screening. Screening can identify persons who suffer from alcoholism, increasing the likelihood that they will receive effective treatment. While this goal may appear to be ambitious, screening can be part of minimal intervention treatment, which may in fact be quite effective, particularly with those who are in the early stages of alcohol abuse, as demonstrated by the well known Malmo project (*38*). In this study, 585 men in their mid to late 40s were identified as ''heavy drinkers'' based on their levels of GGT. At 2- and 4-year followup intervals, GGT levels declined significantly, even for those patients who received only a letter informing them that their liver test showed impairment and advising them to decrease their alcohol consumption. Babor and colleagues summarized the importance of this study by noting that it ''showed that simple intervention based on regular feedback about a biochemical marker can have significant effects on the drinking habits and physical health of a population'' (*39*).

> *'The sensitivity and specificity of self-report measures of alcoholism generally exceed those of biochemical measures, a situation which is not likely to change in the near future.'*

Practicality of screening. From a clinical standpoint, several characteristics are desirable in a screening instrument or series of screening instruments. Instruments should achieve high levels of validity. Careful consideration must be given to establishing an appropriate cut score, given the base rate of alcoholism in that particular setting and the nature of that setting. A well thought-out compromise must be made between sensitivity and specificity. For example, when a physician in a general medical setting can make referrals to a more specialized diagnostic unit, the major concern must be the sensitivity of the instrument, since false positives can be quickly identified at the next stage of the screening process. However, for certain research purposes, and in any legal proceeding where alcoholism is an issue, specificity may be an equal or more important concern.

Other important practical considerations in the selection of screening techniques deal with such matters as cost, the practitioner's need for immediate results, and the kind of decision that needs to be made about referral.

''Covert'' measures. The MacAndrew Scale is less effective than other self-report measures. While it has been criticized, we believe that it is an intriguing instrument because it has some validity, although its item content is restricted to pre-existing MMPI questions. Perhaps a new MacAndrew-like scale with acceptable levels of validity could be created using covert question content, but not limited to the pre-existing questions from the MMPI. For example, items dealing with characteristics that may precede alcoholism (*40, 41*), such as attention deficit disorder, sensation seeking, or impulsivity, might be included. Were this instrument to have acceptable levels of validity, it would be an improvement over other self-report measures. Dissimulation would be more difficult. It might identify the problem earlier, and it might be cheaper and less invasive than biochemical measures.

Choosing between biological and self-report measures. The sensitivity and specificity of self-report measures of alcoholism generally exceed those of biochemical measures, a situation which is not likely to change in the near future. Even if innovative specialized laboratory techniques are refined to the point that they obtain levels of validity comparable to self-report measures, the technology required for quantitative assessments and laboratory procedures probably will require greater sophistication than will be available to most primary health care providers.

However, biochemical measures can provide important new information on alcoholism, including more accurate determinations of severity. Biochemical measures may be useful in identifying those who deny being alcoholic, based on their self-report information. Many clinicians in the field of alcoholism believe that primary health care providers will respond more vigorously to patients with alcoholism if the provider has the results of laboratory tests, rather than a score on a self-report measure. Additionally, biochemical measures are essential to diagnosing alcohol-related pathophysiology.

Among their shortcomings, biochemical markers are less revealing when profound physiological changes have not resulted from alcohol consumption, as is the case with adolescents who have not been drinking long enough to produce such organic change, or with some episodic, or binge, drinkers.

An important research question deals with how self-report and physiological measures might be optimally combined. Some initial work in this area has been reported by Skinner and coworkers (*42*) and by Babor (*7*). Various measures might be combined or instruments could be administered sequentially. Sequencing might take the form of a decision tree or a regression analysis approach.

Research Directions

Important research directions for studies of alcoholism screening are suggested by the preceding discussion. Work on biochemical measures needs to include developing an inexpensive, accurate measure that provides results quickly for the practitioner and the patient. Ideally this measure would indicate not only the presence of alcoholism but also its severity. While measures of alcohol consumption are needed, they need to be correlated with current psychiatric nomenclature diagnoses rather than excessive consumption itself. Efforts are needed to develop a biochemical measure to screen patients early and identify those whose episodic, heavy drinking pattern is associated with risk.

Future efforts on self-report screening instruments might include deriving covert content items so that new instruments are less subject to feigning. Another research goal is to develop self-report measures for use by adolescents. An additional issue which should be investigated is the conceptual meaning of the self-report screening instruments, asking "What are the instruments actually measuring?" Most instruments yield a single score that allows the clinician to make a dichotomous decision of alcoholic *vs.* nonalcoholic. This single domain view of alcoholism differs sharply from most modern conceptualizations of alcoholism, which posit different types of alcoholism. Current self-report measures, however, do not attempt to identify types of alcoholism.

Finally, future research on self-report measure needs to distinguish dependence from severity of the consequences of drinking. Current psychiatric diagnostic systems place major emphases on dependency, yet many self-report screening items deal with harmful consequences. The consequences of drinking are likely to be less valid correlates of alcoholism, since they are "conditioned" by life style and socio-economic variables. For example, if the alcoholic is a self-employed businessman, there will not be criticism from co-workers or a supervisor, and he will not experience such effects of drinking as arriving at work late owing to drinking, which are common items on self-report screening instruments.

Despite the extensive literature contrasting self-report and biochemical measures, we found no studies analyzing the types of errors each measure makes, although several projects contrasted overall accuracy rates. An analysis of errors might help in developing optimal measure-sequencing strategies.

Research on screening is important because it should yield additional means to identify and appropriately refer alcoholics. As more is learned about the nature of alcoholism, more effective means will be developed to identify problems quickly, so that early treatment may be initiated, helping to reduce the financial and emotional cost of this disease.

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Trends in Alcohol-Related Morbidity and Mortality

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Synopsis

Two major trends regarding alcohol use and consequences of alcohol abuse in the United States are showing significant improvement. Continued declines are evident in age-adjusted rates of liver cirrhosis mortality, and per capita alcohol consumption is at its lowest level in 15 years. Two other trends, however, are less clear. After declining in 1982 and continuing through 1984, alcohol-related morbidity—as measured by principal diagnoses listed on short-stay, community hospital discharges—showed a slight increase in 1985. Similarly, after declining every year but one since 1981, alcohol-related motor vehicle fatalities showed a significant increase in 1986.

The downward trends suggest that progress is being made in efforts to reduce alcohol-related deaths and morbidity, but there are no easy explanations for any of the trends. Reductions in liver cirrhosis death rates may reflect coding changes in liver disease categories, less chronic heavy drinking, or better medical care. Lower per capita alcohol consumption may indicate the public's increased awareness of drinking risks or the aging of the U.S. population. Ironically, the recent increase in alcohol-related motor vehicle fatalities may reflect stronger enforcement of drunk driving laws and increased BAC (blood alcohol content) testing.

THE NATIONAL INSTITUTE on Alcohol Abuse and Alcoholism (NIAAA) regularly examines trends in mortality related to liver cirrhosis, alcohol-related fatal traffic accidents, apparent per capita alcohol consumption, and alcohol-related diagnoses in discharges from non-Federal, short-stay community hospitals. These surveillance activities allow the Institute to monitor and analyze both alcohol consumption and key mortality and morbidity indicators that are related to the use and abuse of alcohol.

This paper reports on trends in apparent alcohol consumption and three alcohol problem indicators. The report updates several sections of the epidemiology chapter in the Secretary's "Sixth Special Report to the U.S. Congress on Alcohol and Health" (*1*). However, the information presented is necessarily brief. More detailed information can be obtained from the individual reports (*2–5*), which are available upon request from NIAAA's Alcohol Epidemiologic Data System.

Liver Cirrhosis Mortality

Liver cirrhosis is the ninth leading cause of death in the United States. However, among 25- to 44- and 45- to 64-year-olds, respectively, liver cirrhosis is the sixth and the fifth leading cause of death. According to mortality data from the National Center for Health Statistics (NCHS), liver cirrhosis accounted for 454,722 of the 29.4 million deaths in the United States during the years 1971–85. A total of 26,927 deaths was attributed to liver cirrhosis in 1985, the latest year for which the mortality data are available (*2*).

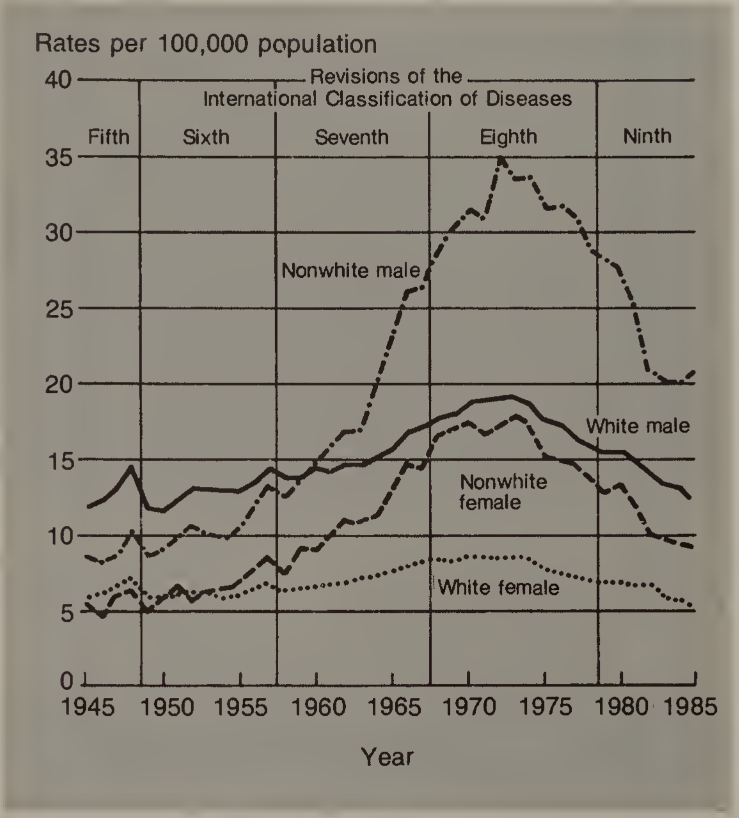
After a sharp increase and then a decline in the years 1945 through 1949, the overall age-adjusted death rate for liver cirrhosis generally increased through the 1950s and 1960s until the rate peaked at 15.0 per 100,000 population in 1973 (data not shown). Since then, the overall cirrhosis death rate has decreased each year, except for a very slight increase in 1980. In 1985, the age-adjusted death rate for liver cirrhosis declined to 9.7 per 100,000 population, a reduction of 35.3 percent since 1973, and the lowest rate in 30 years.

According to race-sex breakdowns of liver cirrhosis death rates, the risk of liver cirrhosis death for males is consistently higher than for females, regardless of race (fig. 1). The most dramatic change in cirrhosis mortality since 1945 occurred among nonwhite males, whose rate increased fourfold by 1973, surpassing the rate for white males in 1960. Rates for white and nonwhite females were virtually identical from 1945 to 1952, but by 1973 the rate for nonwhite females was more than twice the rate for white females.

Declines in cirrhosis mortality, however, have not been consistent across different age-specific race and sex groups (data not shown). Between 1971 and 1985, for example, cirrhosis death rates increased for white and nonwhite women over age 64 and for nonwhite men ages 75 and older. Also, nonwhite men and women ages 25 to 44 years had greater rates of cirrhosis mortality than white men and women in the same age group.

Unlike overall rates for liver cirrhosis, death rates for subcategories of liver cirrhosis have not declined uniformly. Since 1973, specified cirrhosis deaths without mention of alcohol have been declining more rapidly than either unspecified cirrhosis without mention of alcohol or cirrhosis with mention of alcohol. Age-adjusted death rates for alcohol-related cirrhosis were 5.9 per 100,000 in 1971 and 4.4 per 100,000 in 1985, even though proportionate mortality for alcohol-related cirrhosis increased by 12.1 percent. Such discrepancies may be due to true changes or to the use of a greater number of liver cirrhosis subcategories in the International Classifications of Diseases, Ninth Revision (ICD–9) compared to ICD–8 (*2,6*).

Figure 1. Age-adjusted death rates from liver cirrhosis by race and sex, United States, 1945-85



Apparent Per Capita Alcohol Consumption

Each year NIAAA produces estimates of the average per person consumption of alcoholic beverages in gallons of ethanol. The estimates apply to the U.S. population 14 years of age and older, the assumed drinking age population. The per capita estimates of alcohol consumption are based on beverage sales or tax receipt data, or both, from individual States, plus some data from beverage industry sources (*3*).

Trends in apparent per capita alcohol consumption for all alcoholic beverages combined indicate that alcohol consumption rose rapidly from the repeal of Prohibition through 1946, declined in 1947 and 1948, remained relatively stable during the 1950s, and started to rise again in the 1960s and 1970s (data not shown). In the latter period, per capita consumption of all alcoholic beverages

Figure 2. Apparent per capita consumption of alcoholic beverages by beverage type, United States, 1950–86

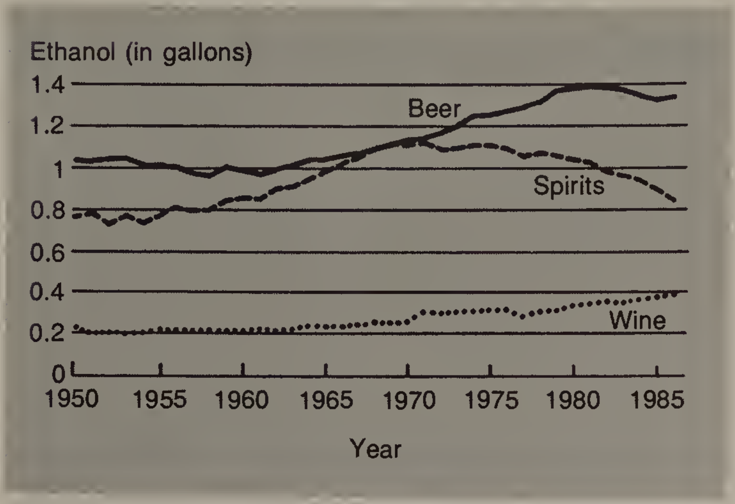
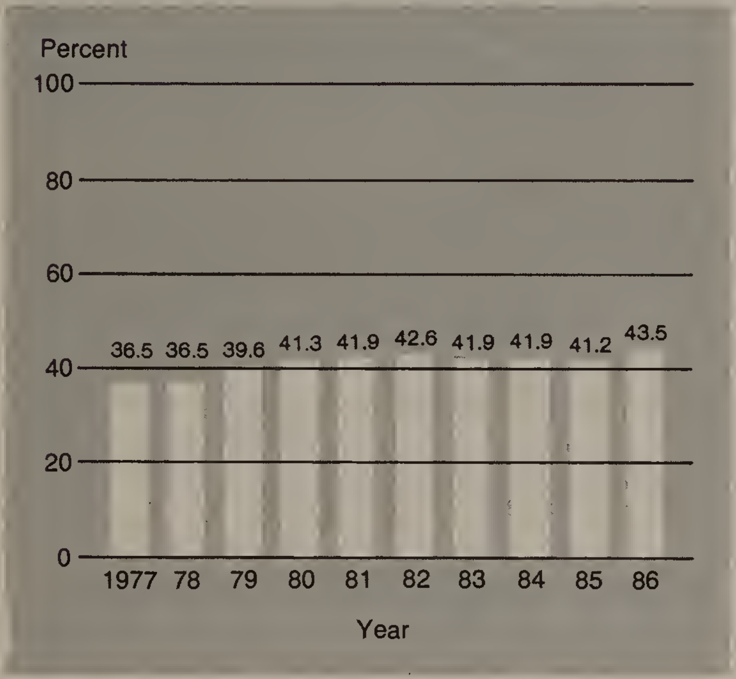


Figure 3. Percent of alcohol-related traffic fatalities among fatal traffic accidents, United States, 1977–86



combined rose 39.4 percent from 1958 to 1981, when the figure peaked at 2.76 gallons. Since then, however, overall per capita consumption has decreased for 5 successive years, ending at 2.58 gallons of ethanol per person in 1986, a decrease of 6.5 percent.

The 1950–86 trends in per capita alcohol consumption by beverage type, on the other hand, show different patterns for beer, wine, and spirits. As shown in figure 2, spirits consumption started to level off or decline around 1970, while beer consumption continued to increase and surpassed spirits consumption by 1970. Per capita spirits consumption declined 20.5 percent between 1978, its peak year, and 1986, the latest year for which the per capita figures have been calculated by NIAAA. Consumption of wine remained relatively stable at around .22 gallons of ethanol per person until 1960. Since then, it has evidenced a rather steady increase to .39 gallons in 1986, an overall increase of 77.3 percent. However, most of the increase in wine consumption since 1984 has been attributed to wine cooler sales (*7*).

Short-Stay Community Hospital Discharges

NIAAA examines alcohol-related morbidity using data from the National Hospital Discharge Survey (NHDS) (*4,8*). The NHDS stratifies non-Federal community hospitals in the United States by size and geographic region and then randomly samples discharges at those hospitals with six or more beds and an average length of stay of less than 30 days. Each hospital discharge record contains from 1 to 7 diagnostic codes that allow examination of principal mention (first-listed) diagnoses and any mention (all-listed) diagnoses. (All-listed or any mention means listed anywhere on the medical record.)

Annual discharges from short-stay community hospitals for persons ages 14 years and older have ranged from 32 to 35 million between 1979 and 1985. Discharges with principal mention of an alcohol-related diagnosis have ranged between 1.7 and 1.9 percent of the total; discharges with any mention of an alcohol-related diagnosis have ranged between 3.0 and 3.5 percent. Obviously, examination of principal diagnoses alone masks morbidity that is associated with alcohol abuse.

Discharge rates per 10,000 population for principal mention and any mention of an alcohol-related diagnosis for 1979 through 1985 are as follows (*4*):

Year	*Principal mention (first-listed)*	*Any mention (all-listed)*
1985	31.2	58.3
1984	30.8	57.6
1983	31.5	57.1
1982	34.4	59.5
1981	37.5	61.8
1980	37.4	61.1
1979	36.1	59.1

Discharges with principal mention of an alcohol-related diagnosis were at a high of 37.5 per 10,000 population in 1981 and decreased steadily to 30.8 per 10,000 population in 1984, a drop of 17.9 percent. In 1985, rates for principal mention of an alcohol-related diagnosis were up slightly from 1984 to 31.2 per 10,000 population but, over the 7 years, rates for principal mention of an alcohol-

Rates per 10,000 population for specific principal diagnoses[1] among hospital discharges with any mention of an alcohol-related diagnosis, United States, 1979–85

Year	*Alcohol dependence syndrome*	*Chronic liver disease and cirrhosis*	*Alcoholic psychoses*	*Nondependent abuse of alcohol*	*Not alcohol-related*
1985	21.1	4.6	3.3	2.1	27.4
1984	21.6	4.4	2.8	1.9	27.0
1983	21.6	5.2	2.5	2.2	25.7
1982	23.4	5.4	3.1	2.6	25.1
1981	25.2	4.2	4.0	2.1	24.5
1980	25.2	6.5	3.7	2.1	23.7
1979	25.6	5.8	2.6	2.2	23.0

[1] See Stinson (*4*) for the components of each of the alcohol-related diagnostic categories.

related diagnosis decreased by 13.6 percent. However, there was no similar decrease in the rates of discharges with any mention of an alcohol-related diagnosis. This disparity suggests that, even though alcohol-related morbidity among hospital discharges has not diminished in recent years, the severity of alcohol-related morbidity may be decreasing.

Also, there has been general consistency in the relative prevalence of specific categories of an alcohol-related principal diagnosis among hospital discharges with any mention of an alcohol-related diagnosis (*4*, see table).

The most prevalent category of alcohol-related principal diagnosis is alcohol dependence syndrome, followed in diminishing order by chronic liver disease and cirrhosis, alcoholic psychoses, and nondependent abuse of alcohol. Among discharges with any mention of an alcohol-related diagnosis, however, the prevalence of nonalcohol-related principal diagnoses has increased steadily from 1979 to 1985, overtaking the prevalence of alcohol dependence syndrome in 1982. This demonstrates again that discharges with a principal alcohol-related diagnosis have decreased proportionately more than discharges with any mention of an alcohol-related diagnosis.

Alcohol-Related Traffic Fatalities

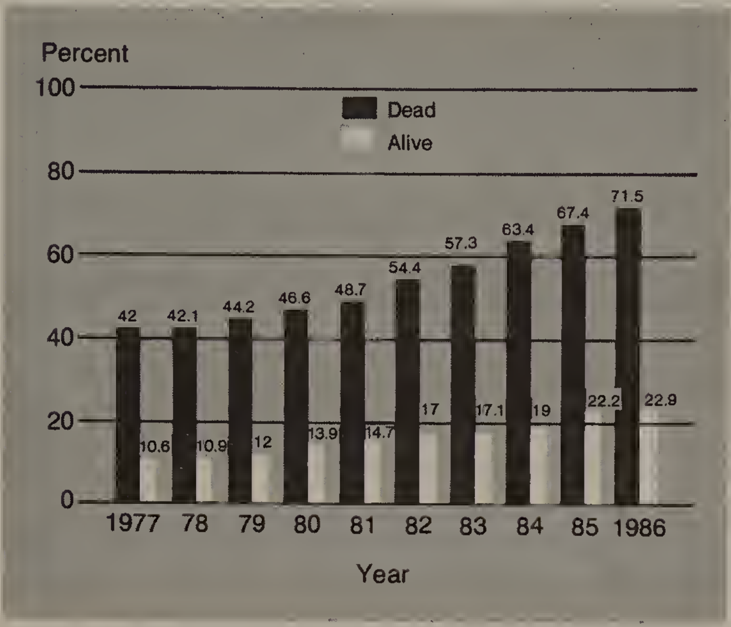
Traffic crashes are the leading cause of death for persons ages 1 to 40 years. Nearly 470,000 traffic fatalities occurred on the nation's highways between 1977 and 1986, and about 191,000 of them (41 percent) were alcohol related (*5*). NIAAA reports annually on proportionate mortality from alcohol-related traffic fatalities using data in the Fatal Accident Reporting System (FARS) of the National Highway Traffic Safety Administration. Alcohol involvement in FARS is determined by investigating officers' reports, tests of blood alcohol content (BAC), or citations for driving under the influence (DUI). NIAAA classifies a fatal crash as alcohol-related if alcohol involvement is reported for at least one of the drivers.

Figure 3 presents the percentage of traffic fatalities attributed to alcohol for the years 1977 through 1986. After increasing each year between 1977 and 1982, the proportion of alcohol-related traffic fatalities among all traffic fatalities remained fairly stable at about 41 percent until 1986. The rate increased to 43.5 percent in 1986, when alcohol-related traffic crashes claimed 20,038 lives. Overall, traffic accident mortality due to alcohol increased 19.2 percent between 1977 and 1986.

Over the same period, there has been a substantial increase in BAC testing among the States. Although some States still have extremely low testing rates, overall BAC testing has increased by 81 percent since 1977. Average BAC testing for both dead and alive drivers was 24.2 percent in 1977 and 43.8 percent in 1986. Figure 4 presents the percentage of driver BAC testing by survival status, that is, dead or alive, for 1977 through 1986.

Young drivers have been and continue to be overrepresented in drinking driver deaths. In 1986, drivers 16 to 24 years old accounted for 38 percent of all drinking driver deaths but only 17 percent of the licensed driver population (data not shown). Also, in 1986 there were 8,483 deaths in crashes involving young drinking drivers (4,532 were the young drivers themselves), up 14 percent from 1985's total of 7,462. Data for 1977–86 indicate that the youngest drivers (16- to 19-year-olds) tend to become involved in fatal accidents at lower BAC levels than do older drivers. This suggests that younger drivers are at an increased risk for alcohol-related fatal accidents because of their lower tolerance to alcohol and their limited driving experience.

Figure 4. Percent of blood alcohol content testing of drivers in fatal traffic accidents according to survival status, United States, 1977–86



Discussion

Specific reasons for the declines in liver cirrhosis mortality are not known, but one reason could be successful alcohol abuse awareness and prevention programs that reduce the population at risk. Another reason could be the changes in definitions, procedures, and coding practices brought about by revisions to the ICD. Grant and her colleagues (*6*) showed that reductions in cirrhosis attributable to alcohol were nearly four times greater with ICD–9 (1978–83 data) than with ICD–8 (1973–78 data). Still another factor influencing the declines could be increased effectiveness of medical treatment for cirrhosis. Powell and Klatskin (*9*) compared survival rates of cirrhosis patients for 1916–36 with those for 1951–63. They found that survival rates in the latter period had increased substantially.

Reasons for the recent declines in apparent per capita alcohol consumption also are unclear. The declines could be attributed to such factors as increased awareness of the risks of heavier drinking (*10*), an increasing preference for beverages with lower alcohol content (*7*), or the aging of the U.S. population, among others. Alcohol consumption tends to decrease with age, and older segments in the U.S. population are increasing relative to the total population. In addition, members of the baby boom generation are starting to enter their mid-40s, near the age when drinking levels start to decline.

Since alcohol-related morbidity and alcohol-related traffic fatalities have both exhibited recent increases after several years of decline, the trends are difficult to interpret. The recent increases may indicate temporary fluctuations in descending trend lines, or the increases could signal either a leveling off or reversals in the rates. Stronger enforcement of drunk driving laws and increased BAC testing, however, have been proposed as part of the reason for the 1986 increase in fatal motor vehicle fatalities (*5*). Stronger enforcement and increased BAC testing may be identifying more alcohol-involved drivers on the nation's highways.

In summary, many factors may have influenced the trends in alcohol-related events, including reductions in per capita alcohol consumption, improved medical management, health promotion efforts, and changes in diagnostic practices of reporting, coding, and classifying morbidity and mortality. Future research should focus on the variability in alcohol problem rates over time. The study of changes in drinking practices (such as level of consumption and binge versus continuous daily drinking) deserves special attention. There is a need to consider variation in rates due to different etiological factors that may be genetic or environmental in origin. Finally, unresolved issues about the reliability and validity of both the alcohol consumption measures and the alcohol problem indicators need to be addressed.

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Assessment of the Potential for Surveillance of Alcohol-Related Casualties Using National Hospital Discharge Survey Data

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This article was adapted from a presentation at the International Research Conference: Statistical Recording Systems of Alcohol Problems, held in Helsinki, Finland, Sept. 14-18, 1987.

Synopsis

It is well known that alcohol abuse is significantly involved in the incidence of casualties (that is, accidents and injuries as they are defined for the purpose of coding diagnoses in the International Classification of Diseases). Thus, a study was conducted of the feasibility of using data from the National Hospital Discharge Survey (NHDS) for the surveillance of alcohol-related casualties. Trends were analyzed over 7 years (1979-85), and results were discussed from three aspects: number and rates for comorbidity of injuries and accidents with alcohol-related diagnoses, percent of alcohol involvement for injuries and accidents, and proportionate morbidity for alcohol-related and nonalcohol-related injuries and accidents.

The incidence of comorbidity and percent of alcohol involvement were found to be relatively low for both accidents and injuries—underreporting being a likely cause. Comorbidity rates over the 7-year period showed no major trends in the rates for injuries that were associated with alcohol use, but the rates for accidents that were associated with alcohol use increased in all but one of the years. Proportionate morbidity as reflected in hospital discharge records with alcohol-related diagnoses showed only small differences by sex and age group (except the 14 to 25 years group) for either injuries or accidents. Only the 25- to 44-year-old group showed a time-trend increase, and that is only for the accident category. For these reasons, we have concluded that data from the NHDS are not currently adequate for use in the surveillance of alcohol-related injuries and accidents.

THE NATIONAL INSTITUTE on Alcohol Abuse and Alcoholism (NIAAA) conducts periodic surveillance of apparent per capita alcohol consumption, mortality from cirrhosis, alcohol-related fatal traffic accidents, and alcohol-related morbidity (*1-4*). This article presents results from an assessment of the potential of the National Hospital Discharge Survey (NHDS) as an appropriate data source for a new area of surveillance, alcohol-related casualties. It is well known that alcohol abuse is significantly involved in the incidence of casualties, which are defined for coding purposes as "accidents" and "injuries" in the Ninth Revision of the International Classification of Diseases, Clinical Modification (ICD-9-CM) (*5, 6*).

The NHDS appears to be a potentially useful source of such data because

- NHDS data are national in scope
- Data collection is ongoing, and new data are published annually
- Definitions, data collection methods, and sam-

'For injuries, males consistently had more than twice as many hospital discharges than females. For accidents, the number of discharges for males was larger than for females, but the difference was not so great.'

pling strategies are consistent from year to year

- The ICD-9-CM (*7*), which is the basis for coding diagnoses in the NHDS, provides codes for accidents and injuries and for certain alcohol-related diagnoses
- The NHDS provides for coding of multiple diagnoses for each discharge record, allowing for examination of comorbidity of alcohol-related and accident or injury diagnoses.

At the same time, certain characteristics of the NHDS might limit its validity for the purpose mentioned. The NHDS includes data only on persons who are admitted to hospitals; therefore, it excludes data on injuries that (*a*) are not treated, (*b*) are treated by a physician on an outpatient basis, (*c*) are treated in an emergency room, and (*d*) are not treated because the injury or accident was fatal. In addition, diagnostic codes available in the ICD-9-CM are useful for identifying alcohol-related conditions that are chronic or are associated with chronic alcohol abuse but are not useful for specifying acute alcohol involvement as a contributor to a hospital episode.

These considerations led to the study described here, which explores morbidity from injuries among the population of persons hospitalized with diagnoses that suggest a history of chronic alcohol abuse.

Methods

Data source. Data for this study come from public-use tapes containing NHDS data for the years 1979 through 1985. This period begins with the first use of the ICD-9-CM and ends with the latest data currently available. The NHDS is conducted on an ongoing basis by the National Center for Health Statistics (NCHS) and is based on the review of discharge records from a sample of non-Federal, short-stay hospitals with six or more beds and an average length of stay of less than 30 days. Each discharge can have up to seven diagnostic codes, allowing for analyses of comorbidity among different diagnoses in the same discharge record. The NHDS does not provide patient identifiers, so an unknown portion of the sampled discharge records may reflect multiple hospital episodes for the same patient(s). Detailed descriptions of the sampling design, data collection procedures, and data collection instruments are published elsewhere (*8*, *9*).

Although the NHDS contains data on discharges for persons of all ages, this study includes data only for discharges of persons ages 14 years and older.

Definitions. The focus of this study is on comorbidity of chronic alcohol-related diagnoses, with diagnoses indicating injuries or accidents. These categories of diagnosis are based on the ICD as follows:

Chronic alcohol-related diagnoses. In this study, hospital discharges were selected as indicating chronic alcohol abuse if they contained diagnoses for any of the following conditions, which are associated with chronic alcohol abuse: alcoholic psychoses (ICD 291), alcohol dependence syndrome (ICD 303, 265.2, 357.5, 425.5, 535.3), cirrhosis of liver (ICD 571, 572.3), and nondependent abuse of alcohol (ICD 305.0).

Injury diagnoses. The ICD-9-CM defines injury diagnoses with codes ranging from 800 through 999. Only ICD 995 (certain adverse effects not elsewhere classified but mostly "due to correct medicinal substances correctly administered") and ICD 996-999 (complications of surgical and medical care not elsewhere classified) were excluded from the injury analyses reported here. These exclusions describe injuries resulting from acts of persons other than the injured person, or are unlikely to be related to the injured person's use of alcohol.

Accident diagnoses. The ICD-9-CM includes a set of "E codes" that are supplementary codes designed to provide information on the external care of injury or poisoning. In this report, references to "accident" diagnoses are intended to be a shorthand for indicating the presence of an E code among the diagnoses appearing on the discharge record. E codes appear on NHDS records with less frequency than injury codes. It is not clear to what extent this results from the fact that a single E code on a record could apply to multiple injury codes on the same record. It may also be that the information required for providing E codes is not always readily available or that physicians feel less of a need to provide such supplementary information. E codes range in value from E800 through E999. With the following exceptions, all available E codes are included and reported as "accidents" in this study: E849 (place of occurrence—for use with selected other E codes), E870–E876 (misadventures to patients during surgical and medical care), E878–E879 (surgical and medical procedures as the cause of abnormal reaction of patient or later complication, without misadventure at the time of procedure), and E930–E949 (drugs, medicinal and biological substances causing adverse effects in therapeutic use). "Place of occurrence" is omitted because it does not code a specific type of accident. The other omissions are for reasons similar to those discussed earlier for injury omissions.

Levels of aggregation. The NHDS data come from a sample of hospital discharges. As with any sample, estimates of the universe based on sample values are subject to error. The reliability of estimates is a function of sample size. Based on NHDS sampling procedures, NCHS has determined that a minimum of 30 unweighted cases is required for even marginally acceptable reliability. Although overall NHDS sample sizes are large (ranging from 160,000 to 185,000 cases per year for persons ages 14 and older for the period covered by this study), the available sample is often too small for reliability when examining morbidity for specific diagnoses. The problem becomes even more severe in analyses of comorbidity, because the analytical cells must contain at least 30 cases for which two separate target diagnoses appear on the same record.

Data presented in this article have been aggregated across diagnostic categories to obtain reliability of estimates for individual years. Care was taken in the analyses to avoid duplicate counting of individual records when multiple injury or accident codes appeared on the same record. The reader is cautioned that the reports of injury and accident diagnoses are not based on mutually exclusive cases; in fact, the accident cases should constitute a subset of the injury cases given the definition of the E code as a supplementary code.

Comorbidity analyses. The analyses of primary interest in this study are those of comorbidity of alcohol-related diagnosis with accident or injury diagnoses—in other words, the incidence with which alcohol-related diagnoses and accident or injury diagnoses appear together for the same discharge record (see fig. 1).

Figure 1. Matrix of the possibilities of comorbidity for an alcohol-related diagnosis in conjunction with an accident (E code) diagnosis

Category	With mention of E code	Without mention of E code	All discharges
With mention of alcohol	A	B	A+B
Without mention of alcohol	C	D	C+D
All discharges	A+C	B+D	A+B+C+D

In figure 1, cell A contains cases where there was both an alcohol-related and an accident diagnosis; this cell contains those cases that show comorbidity for alcohol and accidents. Cell B contains cases with mention of alcohol and no E code, and cell C contains cases with mention of E code and no alcohol. Cell D contains cases where there was no mention of either an alcohol-related or an accident diagnosis.

Analyses of comorbidity are more meaningful if they present results as percents or proportions, rather than as raw numbers. In this study, data are presented using two types of percentage figures: percent of alcohol involvement and proportionate morbidity.

Percent of alcohol involvement. The percent of alcohol involvement shows, for a given diagnosis, the percent of all records with that diagnosis that also had a mention of an alcohol-related diagnosis. Referring to the matrix that is figure 1, this percentage = $100\,(\frac{A}{A+C})$.

Proportionate morbidity. Proportionate morbidity is a measure of the extent to which a particular diagnosis contributes to overall morbidity for any defined group. In this study there are two basic groups for which proportionate morbidity is calculated. Referring to the above matrix, proportionate morbidity for alcohol-related accidents = $100\,(\frac{A}{A+B})$ and proportionate morbidity for nonalcohol-related accidents = $100\,(\frac{C}{C+D})$. The data presented also include alcohol-related proportionate morbidity for discharges separated into groups by sex and by age.

Table 1. Unweighted number of cases with target diagnostic codes, National Hospital Discharge Survey, 1979–85

Year	All diagnoses	All injuries [1]	All accidents [2]	All alcohol	Comorbidity: Injuries [1] and alcohol	Comorbidity: Accidents [2] and alcohol
1979	178,645	18,567	2,948	6,127	534	153
1980	185,402	18,858	3,322	6,443	611	174
1981	188,099	18,807	3,530	6,595	624	197
1982	178,109	17,751	3,594	6,131	569	192
1983	172,011	16,765	3,523	6,017	566	189
1984	160,981	16,326	3,652	5,901	600	216
1985	162,965	16,322	4,024	6,301	644	252

[1] Excludes ICD 995–999.

[2] Excludes E849, E870–E879, and E930–E949.

Table 2. Estimated numbers and rates per 10,000 population for comorbidity of chronic alcohol diagnoses with all injuries and all accidents, 1979–85

Year	Injuries [1]: Number	Injuries [1]: Rate	Accidents [2]: Number	Accidents [2]: Rate
1979	90,867	5.13	26,607	1.50
1980	106,578	5.95	29,991	1.68
1981	105,763	5.81	34,007	1.87
1982	100,002	5.44	33,561	1.82
1983	104,104	5.60	35,570	1.91
1984	111,279	5.91	41,614	2.21
1985	113,668	5.96	45,266	2.38

[1] Excludes ICD 995–999.

[2] Excludes E849, E870–E879, and E930–E949.

Findings

The findings of this study are discussed from three aspects: numbers and rates for comorbidity of injuries and accidents with alcohol-related diagnoses; percent of alcohol involvement for injuries and accidents; and proportionate morbidity for alcohol-related and nonalcohol-related injuries and accidents.

Numbers and rates for comorbidity. One of the purposes of this preliminary study is to assess the frequency with which the target diagnoses (that is, chronic alcohol-related, injury, accident) appear on records in the NHDS. Table 1 presents the number of cases, for each year included in the study, that indicate these target diagnoses, either singly or in combination. It is clear from this table that the NHDS sample size is large. However, the incidence of target diagnoses is relatively small; the incidence of target diagnoses in combination (comorbidity) is substantially smaller. This table also shows that the incidence of E codes in the NHDS is substantially lower than that of injury codes. To some extent this may reflect the fact that a single E code on a discharge record may explain multiple injury codes. However, it appears that E codes may be underutilized in the NHDS.

Table 2 presents the estimated numbers of discharges that show mention of a chronic alcohol-related diagnosis and an injury or accident diagnosis. The estimated numbers are weighted so that they represent estimates of the national incidence of such diagnoses in the population of discharges from short-stay community hospitals with an average length of stay of less than 30 days. For the period 1979–85, these numbers increased for injuries and for accidents, with a more consistent and greater increase for accidents than for injuries. Rates per 10,000 population for discharges with comorbidity for alcohol and injuries or accidents are also presented in table 2. Figure 2 shows the percent change in these population-based rates over the period of study. This figure indicates that rates for accidents associated with alcohol increased in all but one of the years. Over all 7 years, no major trend appears in the rates for injuries associated with alcohol.

Percent of alcohol involvement. One way of examining comorbidity of chronic alcohol-related diagnoses with injuries and accidents is to look at the percent of alcohol involvement for injury and accident diagnoses. The percent of alcohol involvement for injury diagnoses ranged from 2.62 percent in 1979 to 3.57 percent in 1985. Similar figures for accident diagnoses ranged from 4.89 percent in 1979 to 5.95 percent in 1985. Over the 7 years of this study, these figures generally (but not consistently) increased over time.

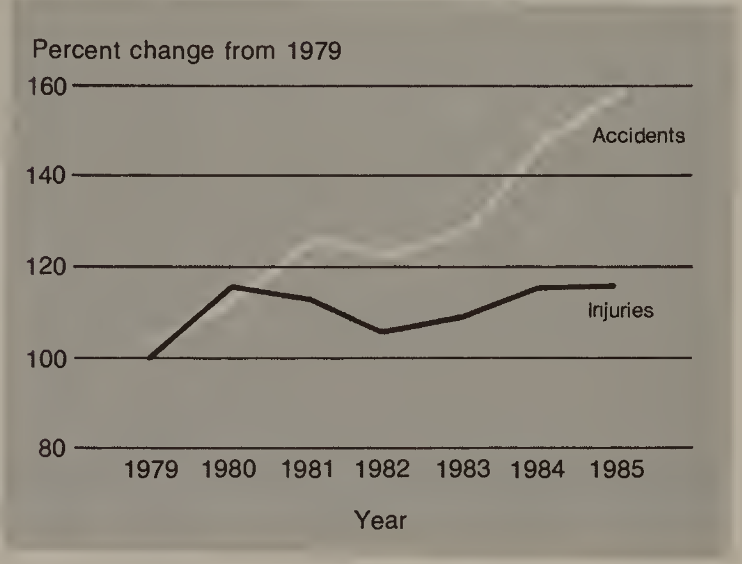
Although it is possible that these apparent changes may reflect actual increases in the involvement of alcohol in injury and accident cases, it is equally likely that they are artifacts of greater attention on the part of physicians to the possibility of alcohol involvement. This second hypothesis is supported by the consistently greater percentage of alcohol involvement in cases with an accident diagnosis. Theoretically, each diagnosis of an injury should be accompanied by a supplementary E code, but this is not seen in the data. The greater apparent association of alcohol with accident codes suggests that those physicians who make the effort to record an E code for injuries also make the effort to record additional, alcohol-related diagnoses.

Proportionate morbidity. Another way to examine comorbidity is to calculate the proportionate morbidity for discharges that mention a chronic alcohol-related diagnosis and for discharges that do not. This statistic provides a measure of the extent to which a particular diagnosis (that is, any injury or accident) contributes to overall morbidity for the group under study. Specifically, proportionate morbidity in this study will indicate the percent of all chronic alcohol-related discharges that also show an injury or accident diagnosis, and the percent of all nonalcohol-related discharges that also show an injury or accident diagnosis. Table 3 shows proportionate morbidity for any injury coded among all nonalcohol-related hospital discharges and all alcohol-related discharges, and proportionate morbidity by sex and age for any injury coded among alcohol-related discharges. Table 3 also shows the same categories of data for any accident. The numerators that provide the basis for the proportionate morbidity data presented in table 3 are contained in table 4.

Consistent with earlier findings, table 3 indicates that injury diagnoses are associated with a larger proportion of alcohol-related and nonalcohol-related discharges than are accident (E code) diagnoses. For injuries, no substantial difference is evident between the proportionate morbidity calculated from alcohol-related discharges and that from nonalcohol-related ones. Nor do there appear to be any consistent trends in proportionate morbidity for injuries.

For accidents, however, table 3 indicates that proportionate morbidity is consistently higher for alcohol-related discharges than for nonalcohol-related discharges. In addition, proportionate morbidity for accidents coded among alcohol-related discharges appears to have increased each year of this study and at a rate greater than is evident for that coded in nonalcohol-related discharges. There is some increase in the coding of accidents for both groups over the period of study. It is not clear to what extent this may be an artifact of an increased awareness of and use of E codes in hospital records abstracted for the NHDS.

Figure 2. Percent change in rates per 10,000 population for comorbidity of alcohol with all injuries and all accidents, 1979–85



We can think of no reason that this increased use of E codes should be greater among patients who have a chronic alcohol-related condition indicated on their hospital records. If patients with chronic alcohol-related conditions are more prone to accidents, and the incidence of accidents for this group were actually increasing over the period of study, we should expect to see similar patterns and differences in proportionate morbidity for injuries. If recent educational efforts have increased the awareness of alcohol-related problems among health professionals, there may be a concomitant tendency for more diligence in seeking an external cause for injuries in patients who exhibit conditions associated with abuse of alcohol. Patients with chronic alcohol-related diagnoses may be more prone to accidents, and this proneness may be increasing over the period of study; but the evidence provided by the analyses we present is not conclusive.

The following discussion of proportionate morbidity will focus only on the group of patients with an alcohol-related diagnosis. Table 3 presents proportionate morbidity for injuries and accidents coded in alcohol-related discharges for males and females separately. For both injuries and accidents, proportionate morbidity calculated from alcohol-related discharges tends to be slightly greater for females than for males, with the differences being

Figure 3. Proportionate morbidity for injuries associated with an alcohol-related diagnosis, by age, 1979–85

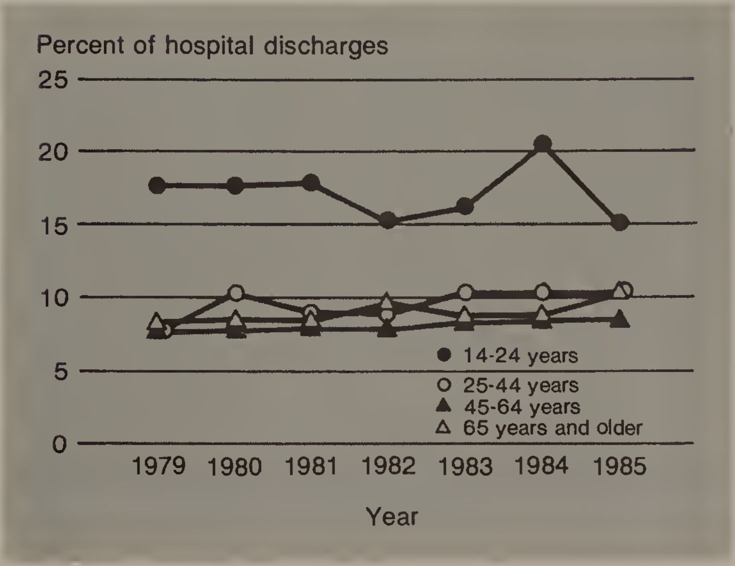
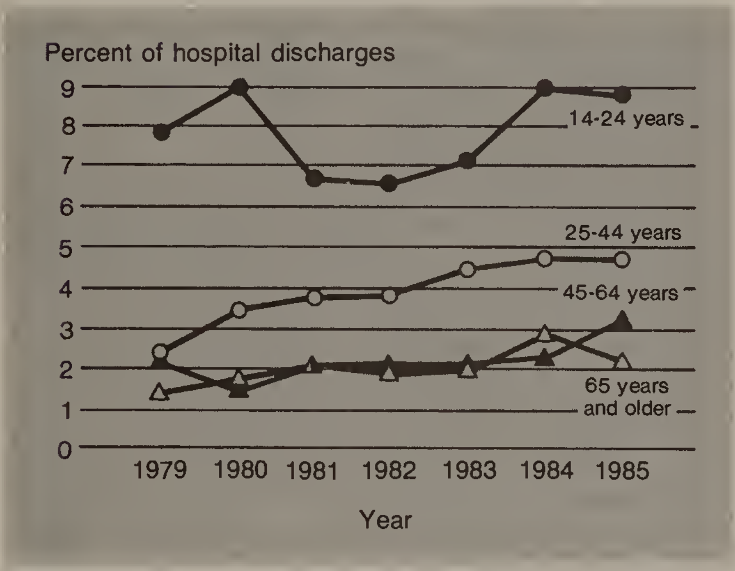


Figure 4. Proportionate morbidity for accidents associated with an alcohol-related diagnosis, by age, 1979–85



larger for accident diagnoses. Reference to table 4 indicates that these proportionate morbidity data are based on more cases for males than for females. For injuries, males consistently had more than twice as many hospital discharges than females. For accidents, the number of discharges for males was larger than for females; but the difference was not so great. In particular, females with chronic alcohol-related diagnoses are more likely than males to have mention of an accident (an E code diagnosis) on their discharge record. Proportionate morbidity for accidents among males with an alcohol-related diagnosis increased over the period of study, approaching levels shown for females in 1984 and 1985.

Figure 3 presents proportionate morbidity for injuries coded among discharges with an alcohol-related diagnosis for different age groups. Although there are no apparent trends in these data, it is clear that the 14–24 years group is consistently more likely to have an injury code than are any of the older age groups included in this study. As can be seen in table 4, the 14–24 years group consistently accounts for approximately one-half to one-third of the injury diagnoses in alcohol-related discharges in comparison with the 25–44 years and 45–64 years groups.

Figure 4 presents proportionate morbidity by age group for accidents reported in discharges with an alcohol-related diagnosis. Again, the age group with the greatest proportionate morbidity is the youngest, 14–24 years. For accidents, however, the 25–44 years group shows greater proportionate morbidity than either of the two older groups; proportionate morbidity for this group appears to be increasing consistently over the period of study. As table 4 shows, this group has the largest number of discharges with an accident diagnosis in all years but 1979. These data suggest that the group of patients 25–44 years old with an alcohol-related diagnosis is increasing its incidence of accidents. We can think of nothing else that would explain this apparent increase for this group that would not also apply to the older age groups in this study.

Implications for Surveillance

The findings reported are of intrinsic interest, because they throw some light on the comorbidity of injuries and accidents with chronic alcohol-related diagnoses. They also raise some significant questions about the use of the E codes defined in the International Classification of Diseases in coding morbidity. In coding mortality, where injury codes are implicated in the cause of death, the supplemental E codes are given precedence in determining the underlying cause of death (*10*). This probably means that E codes are recorded with some care on death certificates.

It appears that E codes do not have the same importance in coding morbidity data. Comparisons of morbidity and mortality data demonstrate this point clearly. Using data presented in table 4, we see that the sum of the estimated numbers of alcohol- and nonalcohol-related hospital discharges showing an injury code is 3,441,771 for 1982. For the same year, the comparable estimate for discharges with an accident code is 689,628. These figures demonstrate that, in the NHDS, E codes are used with about 20 percent of the frequency that injury codes are. In contrast, multiple cause

Table 3. Proportionate morbidity for all injuries [1] and all accidents [2] among hospital discharges, 1979–85

Sex and age group	*1979*	*1980*	*1981*	*1982*	*1983*	*1984*	*1985*
All nonalcohol-related injuries	10.45	10.14	9.89	9.81	9.44	9.89	9.86
Alcohol-related injuries:							
All	8.67	9.73	9.38	9.12	9.79	10.25	10.19
Males	8.53	9.56	9.09	8.92	9.70	10.16	10.20
Females	9.05	10.17	10.09	9.63	9.99	10.44	10.18
14–24 years	17.77	17.86	18.03	15.20	16.25	20.33	15.25
25–44 years	7.81	10.59	9.33	8.87	10.51	10.48	10.52
45–64 years	7.80	7.96	8.07	7.86	8.18	8.51	8.68
65 years and older	8.42	8.77	8.51	9.42	8.72	8.91	10.36
All nonalcohol-related accidents	1.60	1.68	1.75	1.93	1.90	2.13	2.30
Alcohol-related accidents:							
All	2.54	2.74	3.02	3.06	3.34	3.83	4.06
Males	2.05	2.21	2.76	2.59	2.85	3.77	3.91
Females	3.81	4.06	3.64	4.24	4.50	3.97	4.43
14–24 years	7.85	8.94	6.70	6.56	7.17	8.95	8.77
25–44 years	2.33	3.40	3.74	3.82	4.39	4.71	4.68
45–64 years	2.13	1.49	2.02	2.01	2.11	2.26	3.20
65 years and older	1.39	1.70	2.00	1.91	1.94	2.89	2.19

[1] Excludes ICD 995–999.

[2] Excludes E849, E870–E879, and E930–E949.

mortality data published by NCHS indicate that injury codes appear on 194,351 mortality records for 1982. Mentions of an E code on mortality records occur with the following frequencies: 148,852 for E800–E949; 28,438 for E950–E959; 22,679 for E960–E978; and 3,322 for E980–E999 (*11*). Even allowing for some duplication of individual death records in these four E code categories and allowing for the exclusion of some E codes in the morbidity data summarized in table 4, it is clear that the frequency of E codes relative to injury codes is greater in mortality data than in NHDS morbidity data. This finding raises questions about the interpretation of E codes recorded in the NHDS.

The numbers reported in this report, along with certain limitations in the NHDS, indicate that data from the NHDS are not currently adequate for the purpose of providing surveillance of alcohol-related injuries and accidents. This is true for the following reasons:

- The NHDS provides data only on persons admitted to hospitals. Alcohol-related injuries and accidents treated in emergency rooms, or treated by private physicians, or not treated, and fatal alcohol-related injuries and accidents are not necessarily recorded in the NHDS. Such omission means that injuries reflected in the NHDS underestimate the true incidence of injuries in the U.S. population. For example, the NHDS data presented in table 4 indicate that there were an estimated total of 3,457,909 injuries among the population ages 14 years and older in the United States in 1981. In that year, the National Health Interview Survey, a household survey conducted by the NCHS, estimates that there were some 51,652,000 injuries among the U.S. population ages 17 years and older (*12*).
- The data available from the NHDS provide estimates of the numbers of injuries and accidents associated with discharges also showing a diagnosis of a chronic alcohol-related condition. The greater proportionate morbidity for accidents among discharges with an alcohol-related diagnosis may reflect a tendency for those patients to have been using alcohol at the time of the accident, but the data do not support this supposition. The alcohol-related diagnoses included in this study are associated with chronic alcohol abuse and do not implicate the use of alcohol as a proximate cause for any injury or accident recorded.
- The apparent underutilization of E codes in the NHDS means that adequate supplementary information on the circumstances surrounding injuries is not available. From a policy and planning perspective, surveillance of injuries attributed to attempted suicide, motor vehicle accidents, or other categories that place the injury in some context is much more useful than a simple tally of injuries by type (that is, concussion, contusion, open wound) and by location on the body (head, neck, trunk).

These difficulties resolve themselves into two

Table 4. Estimated number of discharges with comorbidity for all injuries[1] and all accidents[2] among hospital discharges, 1979–85

Sex and age group	1979	1980	1981	1982	1983	1984	1985
All nonalcohol-related injuries	3,375,745	3,372,620	3,352,146	3,341,769	3,233,547	3,269,451	3,072,028
Alcohol-related injuries:							
All	90,867	106,578	105,763	100,002	104,104	111,279	113,668
Males	64,751	74,759	72,669	69,795	72,067	76,631	81,505
Females	26,116	31,819	33,094	30,207	32,037	34,648	32,163
14–24 years	14,473	13,765	15,943	14,312	13,494	16,880	13,651
25–44 years	28,839	41,782	38,663	36,380	42,627	44,101	45,896
45–64 years	34,798	36,139	36,569	32,608	32,269	33,273	35,339
65 years and older	12,757	14,892	14,588	16,702	15,714	17,025	18,782
All nonalcohol-related accidents	517,350	558,688	594,410	656,067	651,172	702,702	715,304
Alcohol-related accidents:							
All	26,607	29,991	34,007	33,561	35,570	41,614	45,266
Males	15,599	17,291	22,076	20,271	21,143	28,435	31,283
Females	11,008	12,700	11,931	13,290	14,427	13,179	13,983
14–24 years	6,395	6,889	5,920	6,180	5,952	7,430	7,850
25–44 years	8,599	13,434	15,500	15,677	17,811	19,822	20,406
45–64 years	9,510	6,784	9,151	8,321	8,309	8,843	13,042
65 years and older	2,103	2,884	3,436	3,383	3,498	5,519	3,968

[1] Excludes ICD 995–999.

[2] Excludes E849, E870–879, and E930–E949.

basic issues. First, as it currently exists, the ICD does not have adequate codes for implicating acute alcohol involvement in morbidity or mortality. This issue has been addressed recently by the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) of the Public Health Service, which has recommended the addition of two supplementary alcohol involvement codes for adoption and use in the upcoming 10th revision of the ICD (*13*). The recommended codes are Y92—Evidence of Alcohol Involvement Determined by Blood Alcohol Content and Y93—Evidence of Alcohol Involvement Determined by Level of Intoxication. With the adoption and use of these codes, the ICD and any record-keeping system based on the use of the ICD will be better able to identify events in morbidity and mortality in which the use of alcohol was proximately involved.

Second, for the purposes of surveillance of alcohol-related injuries and accidents, the NHDS does not provide adequate coverage of all events. The authors are unaware of any data collection effort currently operating that provides the required coverage—primarily because accidents and injuries serious enough to require treatment are treated in such a variety of settings that no one system can capture them all. A solution for this second issue will require considerable thought and expenditure of resources in the development of new data collecting systems. A discussion of these issues and a preliminary model for a data collection system for surveillance of alcohol-related casualties was presented at a 1985 Symposium for Statistics on Alcohol-Related Casualties, held in Toronto, Canada (*14*).

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Alcoholism Treatment Service Systems: A Health Services Research Perspective

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Synopsis

This article examines the role of health services research in alcoholism treatment. Alcoholism services research has only recently emerged as a self-defined discipline. Alcoholism services research can be grouped into five classifications: a) *descriptive studies of resources for alcoholism treatment and of the use or cost of these services,* b) *estimates of the need or demand for alcohol services in the population or in particular subpopulations,* c) *studies of the costs or cost-effectiveness of alcoholism treatment or of alternative treatments,* d) *studies of the possible "cost-offsets" of treating alcoholism, and* e) *studies that examine strategies for financing and reimbursement for alcoholism treatment. Research is needed to determine how alcoholism treatment services are now delivered, who uses these services, how treatment setting and organization affect service delivery, who pays for alcoholism treatment, and how reimbursement policies affect the delivery of alcoholism services. Research on large-scale social issues is also needed, such as the effects of warning labels appearing on alcoholic beverage containers or estimates of the overall cost to society of alcohol abuse.*

RECENTLY, AN ADVISORY BOARD created by Congress to assess national needs for alcohol, drug abuse, and mental health services recommended that research on treatment services should be declared a priority area (*1*). At the present time, research on alcohol service systems receives a relatively low priority within the Federal Government and in the alcoholism field generally.

A number of important health services research studies have been conducted in the alcoholism treatment area (*2*), many supported by the National Institute on Alcohol Abuse and Alcoholism (NIAAA). Even so, the field lacks a well-defined sense of goals and methods. This article examines the concept of health services research as it has been used in other health areas and relates it to alcoholism treatment issues. Included are some possible directions for future research.

History and Definition

The importance of health services research was formally recognized by Congress in 1974, when the Public Health Services Act, one of the principal acts of Congress providing legislative authority for Federal health activities, was amended to create the National Center for Health Services Research (NCHSR) within the Public Health Service (*3*).

Many definitions of health services research stress its hybrid nature. Health services research combines concepts and techniques from economics, medicine, sociology, psychology, epidemiology, business administration, and a number of other disciplines.

Other definitions stress the subject of the research. In a dictionary of health care terminology prepared in 1976 by the House Subcommittee on Health and the Environment, health services research was defined as "research concerned with the organization, financing, administration, effects, or other aspects of health services; rather than with human biology or disease and its prevention, diagnosis, and treatment" (*4*). In 1979, the Institute of Medicine of the National Academy of Sciences, in a report on health services research in the Federal Government, defined health services research as "inquiry to produce knowledge about the structure, processes or effects of personal health services"(*5*). According to the report, a "personal health service" is any "interaction between a provider of health services and a client for the purpose of promoting the health of the client."

The Institute of Medicine report offered two criteria necessary to define research as health services research. First, the research must deal with some features of the structure, processes, or effects of personal health service. Second, at least one feature must be related to a conceptual framework other than that of contemporary applied biomedical science (*5*).

In the years since its recognition as a discipline, health services research has continued to expand both in the public and the private sector. In the 1970s, much of the impetus for the growth of the field came from the debate over national health insurance. In the 1980s, research has been most often stimulated by public and private initiatives to contain health care costs. In 1983, a health services research professional association, the Association for Health Services Research (AHSR), was founded. Based in Washington, DC, AHSR serves as an active lobby in Congress on behalf of health services research (*6*).

Types of Health Services Research

In addition to defining health services research, the Institute of Medicine report (*5*) gave an excellent description of the kinds of research included in the field. The report's authors noted that studies of health services may address problems at any one of the following four levels.

- Clinical level. Although methodologically similar to biomedical studies that are clinically oriented, health services research studies at the clinical level take into account nonmedical considerations that affect outcomes (such as setting and provider characteristics) and also address a broad range of outcome criteria (such as patient satisfaction or treatment costs).
- Institutional level. Although institutional studies in health services research also may share many of the concerns of clinically oriented research, they focus more on organizational and administrative features of the settings where services are delivered. Emphasis may be on how the organization of services affects outcomes (such as quality of care or cost) or on how patient characteristics (such as income or insurer) affect the setting.
- Systems level. Studies at the systems level examine interrelationships among various aspects of the health care system. Systems studies, for example, may attempt to understand how financing mechanisms, the organization of health care services, the demand for health care, and health care expenditures are related.
- Environmental level. Environmental studies seek to understand the circumstances and events in the larger social, political, and economic contexts that shape the health services system and define its functions in relation to the overall social system.

The report noted that the collection of data to administer programs does not constitute health services research unless analysis of these data is directed toward answering a question that applies to other programs or organizations as well. This qualification does not necessarily exclude descriptive research as an important kind of health services research. Data such as statistics on the use of health care services or estimates of national health care expenditures can identify trends and variations that raise theoretical and policy questions. These questions may then help guide analytic research. Analytic research in turn attempts to answer cause-effect questions or to make projections.

Mental Health Services Research

Mental health services research is a subcategory of health services research. It has its own history as a specialized research discipline with its own professional identity. The field was substantially affected in its early years by the community mental health movement of the 1960s, which generated both issues and funding for mental health services research, but the National Institute of Mental Health has been collecting data on mental institutions as far back as the 1840s (*7*). As the delivery of mental health services continued to move out of State mental hospital settings and into the primary care sector, the issues of concern to mental health researchers have become similar to those of researchers working in the general health care sector (*8*). The Association for Health Services Research now publishes a newsletter devoted exclusively to mental health research.

Mental health services research, like health services research generally, has been strongly influenced in recent years by public and private initiatives to contain health care costs and to develop appropriate financing and reimbursement strategies. Other priority areas, at least as indicated by research support from the National Institute of Mental Health, include the provision of mental health care in the primary sector and the organization and delivery of care for the chronically mentally ill (*9*).

Alcohol Services Research

Alcohol services research has a shorter history than mental health services research as a self-defined discipline. Nonetheless, the same trends in health care and health care financing that have influenced research in physical and mental health services research are also important in relation to research on alcohol services research. Indications are now appearing that alcohol services research is being defined as a field in its own right. In 1987, one of the panels at the most recent National Conference of the Alcohol and Drug Problems Association was titled "Alcoholism Treatment Service System Research."

Most alcohol services research fits into one of five major categories:

- Descriptive studies of resources for alcoholism treatment and of the use or cost of these resources.
- Estimates of the need or demand for alcohol services in the population or in particular subpopulations.
- Studies of the costs or cost-effectiveness of alcohol treatment or of alternative treatments.
- Studies of the possible "cost-offsets" of treating alcoholism.
- Studies of possible strategies for financing and reimbursement for alcoholism treatment or assessments of the impact of alternative strategies on the organization, cost, delivery, quality, availability, or outcomes of alcoholism treatment.

> *'Only about 15 percent of individuals with diagnosable alcohol problems receive alcohol treatment services.'*

The following discussion delineates these areas and points to some of the limitations of existing research in each area.

Descriptive studies. Assembling descriptive data on alcoholism treatment resources and their use is an important aspect of alcohol services research. The National Drug and Alcoholism Treatment Utilization Survey (NDATUS), jointly sponsored by NIAAA and the National Institute on Drug Abuse (NIDA), is a major vehicle for collecting periodic data on the capacities, utilization rates, funding sources, staffing patterns, and client and staff demographic characteristics of alcohol and drug treatment facilities (*10*).

Many alcoholism treatment services are provided outside of specialized alcoholism treatment facilities. Alcoholics admitted for alcohol problems to short-term hospitals or for medical complications related to their alcoholism are often treated in hospitals with no alcoholism or chemical dependency treatment units (*11*). Little is known about the care provided to alcoholics in these settings. Still less is known about "minimal" or informal interventions that may be provided in primary care or in nonmedical settings.

A comprehensive typology is needed for alcoholism treatment services and settings and a description of the frequency with which various services are provided in different settings. The NDATUS survey produces limited information on services by setting, but many services are not identified because NDATUS collects data only on facilities offering specialized alcoholism or chemical dependency treatment. Data on treatment provided outside these specialized facilities, including information on the use of Alcoholics Anonymous (AA), would improve our ability to describe the alcoholism treatment service system. Such data would also assist clinical researchers in their attempts to characterize treatments for the purpose of studying the relative effectiveness of different treatment modalities for different kinds of patients.

'Research to date has generally failed to demonstrate that any one treatment or treatment setting is better than another . . . One issue of particular concern is the relative cost-effectiveness of inpatient versus outpatient treatment.'

Estimates of Need

Existing research suggests that the need for alcoholism services in the population is far greater than might be assumed on the basis of current patterns of use. Only about 15 percent of individuals with diagnosable alcohol problems receive alcoholism treatment services (*11*).

Some populations are at particular risk for alcohol problems and may also be high users of other kinds of health care services. Relatively little is known, for example, about the needs and patterns of health care use of persons with combined alcohol, drug, or mental health diagnoses (for example, alcohol and drug, alcohol and mental disorder). Research is also needed on the general health care costs and use patterns of individuals with alcohol problems.

Cost-Effectiveness

Cost-effectiveness research in alcoholism treatment addresses the question of how efficient or how costly a particular program or type of program is in producing a particular outcome, usually abstinence. The findings of such research are important for policy and planning decisions concerning the financing, organization, and delivery of alcoholism treatment services. They are also valuable as a guide to appropriate reimbursement strategies for Federal and State governments as well as for employers and private insurers.

Probably because of their clinical focus, studies of the outcomes of alcoholism treatment have rarely measured the cost of treatment. In a paper prepared for an NIAAA Ad Hoc Scientific Advisory Board meeting on alcoholism treatment, Putnam (*12*) noted that those studies that do compare treatment costs typically do not include an outcome measure of drinking behavior. Instead, outcomes are defined in terms of reduced use of services, most often health care services.

A major problem in research on the cost-effectiveness of alcoholism treatment is highlighted in a report prepared by the Office of Technology Assessment (OTA) in 1983 (*13*). Research to date has generally failed to demonstrate that any one treatment or treatment setting is better than another. Although it is possible to compare treatments with respect to cost, it is difficult to develop a measure of relative effectiveness. The OTA report concluded that there is some evidence to support the conclusion that alcoholism treatment in general is cost-effective, but there is little evidence for concluding that one kind of treatment is more cost-effective than another. One issue of particular concern is the relative cost-effectiveness of inpatient versus outpatient treatment.

Cost-Offsets

The primary question asked by authors of cost-offset studies is whether the benefits of alcoholism treatment outweigh the costs of treatment. The question of costs and benefits is somewhat different from the question of cost-effectiveness. For instance, the most cost-effective treatment for alcoholism might still be quite expensive in absolute terms. It would be cost-beneficial, then, only if its cost were offset by cost reductions in another area. Cost-effectiveness studies do not address this issue because they only show the cost of treatment; they do not measure the "degree of cure" in cost terms. They also do not compare the overall costs of treating alcoholism with the costs of not treating alcoholism.

Ideally, cost-offset studies compare the dollar cost of treatment with the dollar costs of the untreated condition. Unfortunately, the cost of treatment is often difficult to determine from available data, and the cost of the untreated condition is even more difficult to measure. Proxy measures of cost, such as number of physician visits or days of work missed, are often used instead.

For example, some cost-offset studies have examined the effects of alcoholism treatment on the use of medical care services (*14–16*). Alcoholics and their families tend to use a disproportionate amount of medical care. The researchers have found that alcoholism treatment is typically followed by reduced use of medical care services on the part of alcoholics and, in some cases, on the part of family members. Putnam (*12*) points to a need for more studies that incorporate both cost and use data. She also argues that outcome should be directly measured in these studies. Because the assumption that the treatment has actually worked is rarely addressed in cost-offset literature, the research does not establish that the alcoholism treatment accounted for the reduced use of medical care services.

Another point made by Putnam is that it is important to understand precisely how the use of medical care services changes after alcoholism treatment. Treated alcoholics may simply be substituting other services (such as mental health and counseling services) for the medical care they may have been using inappropriately before treatment. If so, it is their pattern of use, not their health, that has changed. Another possibility is that families may not use health care services after alcoholism treatment because the costs of the treatment may have strained their budget. In this case, their reduced use of health care service would be a negative consequence of treatment. Still another possibility is that treated alcoholics and their families may become more sensitive to general health care needs and therefore may seek care sooner, when the condition is easier and less costly to treat. Research is needed to distinguish among these possibilities.

Another problem with the cost-offset research is that few studies cover a sufficient number of years before and after treatment to guarantee that long-term use patterns are being revealed (rather than the immediate effects of the crisis that may have precipitated the alcoholic into treatment). Some studies have investigated the overall cost to society of untreated alcoholism and have related it to treatment costs. Medical care costs account only for a part of the total costs of untreated alcoholism. Other social costs include reduced productivity, motor vehicle and other property damage, incarceration, fetal alcohol syndrome, child and spouse abuse, and years of productive life and income lost, as well as such noneconomic costs as family disruption, emotional difficulties, and lower work morale. The estimated cost of alcoholism in 1983 was $117 billion (*17*).

Putnam (*12*) points out that employer-based studies, especially when the employers are large corporations and are self-insured or have a large medical department, offer a rich opportunity for studying social costs and cost-offsets. Because they are relatively closed systems, large corporations can provide data on the behavior of treated and untreated alcoholic employees in a number of areas related to the social costs of untreated alcoholism and to treatment outcomes. Two such large-scale employer-based studies are currently being funded by NIAAA: "A Randomized Trial of Worksite Alcoholism Treatments," directed by Diana C. Walsh (R01 AA06461), and "Health Care Costs for Employed Alcoholics in Treatment," directed by Harold D. Holder (R01 AA06248). It is also important to include unemployed populations in estimates of the overall costs of untreated alcoholism, although these groups are more difficult to study (*12*).

Financing and Reimbursement

Financing refers to the general questions of who pays for alcoholism treatment services and how these funds are distributed by third parties. Reimbursement refers specifically to the question of how and for what services the providers are paid by third-party payers.

Federal, State, and local governments are major sources of financing for alcoholism treatment services (*2*) but coverage for alcoholism treatment in employer-based insurance policies has been expanding rapidly. According to the Bureau of Labor Statistics, the percentage of employees with health coverage for alcoholism treatment increased from 38 percent in 1981 to 70 percent in 1986 (*18*).

Descriptive data on the financing of alcoholism treatment services are needed to assess the effects of current financing trends. These trends include the change to Federal block grant funding, the increase in private insurance coverage for alcoholism services, the increase in private ownership of alcoholism treatment facilities, and changes in reimbursement policy (*1*). Important changes in reimbursement include the rise of health maintenance organizations and preferred provider organizations, as well as Medicare's prospective payment system based on the diagnosis-related groups (DRG).

Research is also needed to assess access on adequacy of insurance coverage for alcoholism treatment in the population.

Key Questions

The following list summarizes key questions for alcohol services research and suggests data that could be used to address them:

- How are alcoholism treatment services now delivered? What does the alcoholism treatment service system look like, and what services are provided in what settings? What are the characteristics of the clients who use these services, and how does cost vary from one setting to another?

- Who uses alcoholism treatment services? How do clients in different treatment settings and with different kinds of insurance differ from one another in their treatment needs and in their other characteristics (income, social stability, education, etc.)? How do patients receiving alcoholism treatment differ from individuals with alcohol problems who do not receive treatment?
- How does the organization or setting in which a service or treatment is provided affect its quality, effectiveness, cost, and accessibility?
- Who pays for alcoholism treatment? How are the costs and benefits of alcoholism treatment distributed in society?
- How do various reimbursement policies influence the supply and organization of alcoholism treatment services and the demand for these services?

Data bearing on these questions can be gathered at each of the four levels identified by the Institute of Medicine in its report on health services research (5).

At the clinical level, it would be extremely useful if studies of the effectiveness of treatment collected data on treatment costs as well as treatment outcomes. These data would permit cost-effectiveness analyses to be carried out even when cost factors were not the direct focus of a study.

At the institutional level, a number of data bases are available that provide data on services utilization in particular settings or by particular populations, such as HMOs, large self-insured corporations, the military, the Veterans Administration, Medicare, and private insurers. These data bases could be used for studies of economic factors in the delivery of alcoholism treatment services or the influence of program organization on access to, or cost of, care.

Data on whole health care systems could be obtained from State data bases, from NDATUS, CHAMPUS, and the Epidemiological Catchment Area data compiled by the National Institute of Mental Health, and from such surveys as the National Ambulatory Medical Care Survey, conducted by the National Center for Health Statistics.

Attempts to obtain data at the environmental level, relating alcoholism treatment to the larger health care system and to larger social and political factors, such as the level of economic development in society, might be premature at this time, although international cooperative studies might be one source of such data. For example, the AMETHYST project, sponsored by NIAAA and the World Health Organization, is studying the effectiveness of a low-cost screening and early intervention technique in several different countries. These data might be useful for some environmental analyses. Other large-scale social issues include the question of the effects of warning labels on alcoholic beverage containers or estimates of the overall cost to society of alcohol abuse and alcoholism.

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Alcohol Problem Resources and Services in State Supported Programs, FY 1987

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This paper consists primarily of excerpts from a report entitled "State Resources and Services Related to Alcohol and Drug Abuse Problems, Fiscal Year 1987—An Analysis of State Alcohol and Drug Abuse Profile Data," which was published in August 1988. All of the data for the report were voluntarily contributed by the State Alcohol and Drug Agencies from all 50 States, the District of Columbia, Guam, and Puerto Rico.

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Synopsis

During fiscal year 1987, expenditures for alcohol and drug abuse services in facilities receiving at least some funds from State alcohol and drug agencies totaled $1,809,749,013. Of this total, approximately 51.1 percent was contributed by State governments, 17.9 percent by the Federal Government, 9.1 percent by county or local agencies, and 21.9 percent by other sources (for example, private health insurance).

Approximately 76.5 percent of the funds was expended for treatment services, 12.6 percent for prevention services, and 10.9 percent for other services (for example, administration, research, training). Between fiscal years 1985 and 1987, total expenditures increased 31.2 percent, although great variability existed among States, with some undergoing significant cuts.

The total number of alcohol and drug treatment units that received State funds was 6,632. During fiscal year 1987, admissions for alcoholism treatment in these State-supported facilities totaled 1,317,473. Most admissions were to a nonhospital environment (84.6 percent) and were for outpatient care (44.9 percent).

Of the total number of admissions, 76.2 percent were men and 19.8 percent women; the sex of 4.0 percent was not reported. With regard to age, 27.4 percent were 25–34, 21.7 percent were 35–44, 10.7 percent were 21–24, and 4.1 percent were under 18.

In terms of race or ethnicity, 69.7 percent of those admitted were white, 15.6 percent black, 5.5 percent Hispanic, 3.6 percent Native American, 0.2 percent Asian or Pacific Islander, 0.3 percent others, and 5.2 percent not reported.

Compared with the 1,317,473 admissions for alcoholism, the combined total of all other drug admissions was only 450,553. The highest numbers of other drug admissions were 98,549 for heroin, 84,707 for cocaine, and 63,740 for marijuana/hashish. Also, compared with drug treatment admissions, those admitted for alcoholism are more likely to be male, white, and older.

ALCOHOL AND DRUG ABUSE dependency constitute major public health problems for the nation. During 1983, the most recent year for which cost data are available, the economic costs of these problems totaled more than $176 billion (*1*). These enormous problems must be addressed at all levels of government. At the Federal level, the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), the National Institute on Drug Abuse (NIDA), and the Office for Substance Abuse Prevention (OSAP) have the responsibility to provide national leadership on alcohol and drug issues. A significant portion of this responsibility focuses on the task of monitoring various indicators of alcohol and drug abuse, including information on treatment and prevention services and funding resources.

At the State level, the State alcohol and drug agencies have administrative responsibility for the allocation and effective use of Federal and State funds specifically targeted for alcohol and drug treatment and prevention services. To carry out these tasks effectively and efficiently, each State agency collects relevant information on needs, services, and resources to assist ongoing planning, monitoring, and service delivery functions.

Before 1982, NIAAA and NIDA were the repostories of detailed information from States and programs on federally funded alcohol and drug treatment and prevention services and clients. Data were reported by the States or individual programs, or both, as a condition of receipt of the Federal alcohol and drug formula grant and project grant funds. However, when the Alcohol, Drug Abuse, and Mental Health Services Block Grant was authorized by Public Law 97-35 in 1981, the requirement for the provision of detailed data from the States and individual programs was no longer mandated.

Nevertheless, the continued importance of and need for national data on alcohol and drug treatment and prevention programs, services, and clients were recognized at both the Federal and State levels. The Senate Committee on Labor and Human Resources included language in its report on the Alcohol and Drug Abuse Amendments of 1983 that referred to data collection as "an important national leadership responsibility of the Institutes." The committee specifically encouraged the Institutes to acquire "alcoholism and drug program data from information systems in each State." Congress eventually directed the Secretary of Health and Human Services, through the Administrator of ADAMAHA, to

> . . . conduct data collection activities with respect to such programs, including data collection activities concerning the types of alcoholism, alcohol abuse, drug abuse and mental health treatment and prevention activities conducted under such part, the number and types of individuals receiving services under such programs and activities, and the sources of funding (other than funding provided under such part) for such programs and activities (Sec. 1920, 42 U.S.C. 300 x).

Part B, title XIX, of the Public Health Services Act further requires that

> The Secretary, in consultation with appropriate national organizations, shall develop model criteria and forms for the collection of data and information with respect to services provided under this part in order to enable States to share uniform data and information with respect to the provision of such services.

To meet these congressional mandates, the Federal Government has sought to maintain minimal data that are accurate and updated regularly. Since 1982, the National Association of State Alcohol and Drug Abuse Directors (NASADAD) has collected, analyzed, and disseminated uniform information provided by the States on alcohol and drug abuse treatment and prevention resources and clients. These activities have been supported by NIAAA, NIDA, and the States. The States' willingness to provide NASADAD with information on alcohol and drug treatment and prevention services, resources, and clients is evidenced by successful previous contract efforts that included State data from fiscal years 1983-86 (*2-6*).

NASADAD worked with ADAMHA, the Institutes, and the States to assess, define, and provide information existing at the State level on alcohol and drug abuse services, programs, resources, and needs. The major tasks were the definition and collection of information in a uniform format from NASADAD members, the analysis of the data submitted by each State, the development of meaningful comparisons of data across States and over time, the provision of a comprehensive report on the findings, and the conduct of two special studies per year. Data from fiscal year 1987 were analyzed and compared with data from previous fiscal years.

Study Purpose and Methodology

The overall purpose of this report was to ensure the continued availability of selected service and resource information, including financial, program, and client data, from existing State sources throughout the United States and the Territories. These data are to assist NIAAA and NIDA in assessing the type of treatment and prevention resources and services provided to persons throughout the country who are dependent upon or abusing alcohol and other drugs.

The major study objectives were to provide continued support for the implementation of a joint Federal-State data strategy, through collaboration on the State Alcohol and Drug Abuse Profile and the National Drug and Alcohol Treatment Utilization Survey; to compile annually secondary data relating to alcohol and drug abuse services, clients, and resources; to automate the editing, storage, and analysis of data acquired in prior and current fiscal years; and to aggregate and analyze the data, including the development of comparisons and analyses within and across States.

The overall study methodology included the design of data acquisition and analysis plans, the development of support materials and procedures, the implementation of data acquisition and analysis, and the preparation of numerous project reports.

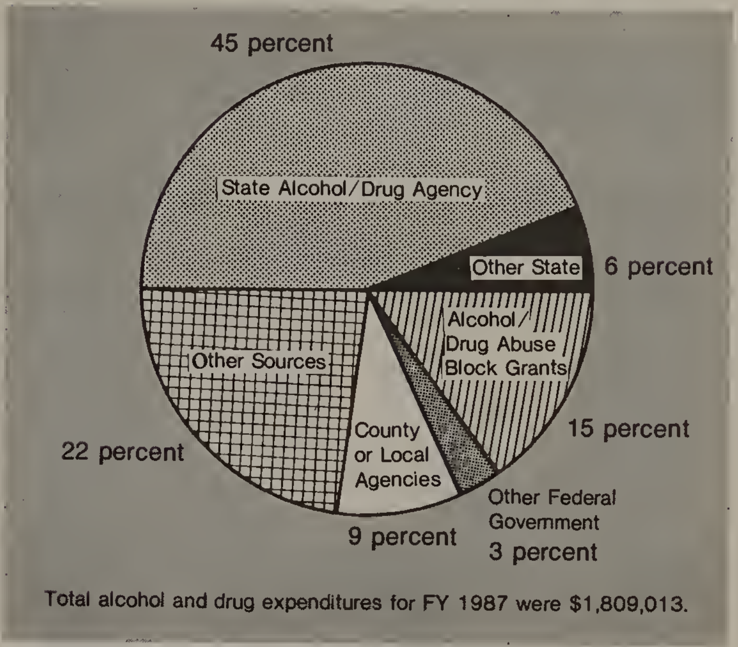
After obtaining input from State and Institute representatives, NASADAD staff began collecting data in October 1987 from the State alcohol and drug agency directors. Followup letters and telephone calls were made to directors who had not submitted information within the requested time.

The directors of the State alcohol and drug agencies from 50 States, the District of Columbia, Guam, and Puerto Rico voluntarily submitted the requested information. The data received are summarized and analyzed in the remaining sections of this report. Each State director was requested to review and verify the accuracy of all information from his or her State.

Funding of Alcohol and Drug Services

Data are presented on total expenditures for alcohol and drug services by source of funding and type of program activity within the State for that State's fiscal year 1987. These data have a number of limitations. One major qualification is that the States were asked to report total expenditures for "*only those programs which received at least some funds administered by the State Alcohol/Drug Agency during the State's Fiscal Year 1987.*" The data do not include information on programs that did not receive funding from the State alcohol and drug agency (for example, most, if not all, private for-profit programs, some private not-for-profit programs, and some public programs). As a result, the overall fiscal estimates are conservative and, to varying degrees, underestimate funding by other departments of State governments, by Federal agencies such as the Veterans Administration, and by privately supported alcohol and drug abuse treatment and prevention programs.

Figure 1. Expenditures for State-supported alcohol and drug abuse services by funding source for Fiscal Year 1987



NOTE: The "Other Sources" category includes funding from sources such as client fees, court fines, and reimbursements from private health insurance.

SOURCE: State Alcohol and Drug Abuse Profile, FY 1987; data are included for "only those programs which received at least some funds administered by the State Alcohol/Drug Agency during the State's Fiscal Year 1987."

The financial and related data collected from States for fiscal year 1987 are organized into three major subsections: financial expenditures by State and funding source, financial expenditures by type of program activity, and total number and percentage of treatment units that received funds administered by the State alcohol and drug agency.

Expenditures by source of funds. Individual and total expenditures are categorized by State, by funding source, and by comparison of fiscal year 1985 with 1987, including State alcohol and drug agency funds, other State funds, the alcohol and drug portion of the Alcohol, Drug Abuse, and Mental Health Services and the Emergency Alcohol and Drug Treatment and Rehabilitation Block Grants, other Federal funds, county and local funds, and funds from other sources (table 1).

The total expenditures for all 50 States, the District of Columbia, Guam, and Puerto Rico for alcohol and drug services in those programs receiving at least some State-administered funds during each State's fiscal year 1987 were $1,809,749,013.

Table 1. Expenditures for State-supported alcohol and drug abuse services by State and by funding source for fiscal year 1987 and comparision of fiscal years 1985 to 1987

State	State alcohol-drug agency	Other State agency	Alcohol Drug Abuse Block grants	Other Federal Government	County or local agencies	Other sources	Grand total	Percent change 1985 to 1987
Alabama	2,695,411	N/A	3,927,275	680,414	N/A	N/A	7,303,100	23.5
Alaska	12,661,000	715,000	2,064,100	2,538,000	2,150,000	1,400,000	21,528,100	10.3
Arizona	8,956,984	574,541	5,291,161	N/A	N/A	7,554,951	[1,2] 22,377,637	10.7
Arkansas	2,615,596	0	2,439,726	984,279	615,326	0	6,654,927	23.2
California	78,255,000	1,497,000	34,051,000	3,619,000	34,534,000	69,116,000	[1] 221,072,000	9.5
Colorado	11,590,676	949,871	3,834,124	263,674	3,904,702	4,903,171	25,446,218	56.9
Connecticut	17,551,722	0	4,449,498	2,438,193	0	16,026,802	40,466,215	49.4
Delaware	2,719,750	162,056	1,097,252	19,245	0	8,427	4,006,730	6.6
District of Columbia	128,468	23,242,311	1,775,200	206,455	N/A	1,225,751	26,579,185	40.6
Florida	33,801,984	1,095,000	20,942,288	289,138	18,653,311	N/A	74,781,721	74.3
Georgia	24,433,091	N/A	3,925,110	156,211	701,268	2,930,194	32,145,874	35.1
Guam	N/A	N/A	209,937	N/A	41,956	N/A	251,893	. . .
Hawaii	1,872,722	N/A	1,243,880	N/A	20,347	1,212,309	4,349,258	18.4
Idaho	1,727,100	N/A	642,800	32,300	N/A	3,700	2,405,900	−14.8
Illinois	52,939,400	100,000	11,456,300	1,014,200	N/A	0	65,509,900	38.3
Indiana	4,957,827	10,879,167	2,798,747	2,675,225	N/A	N/A	21,310,966	20.5
Iowa	7,504,361	1,151,507	2,319,161	192,194	1,315,417	2,155,263	14,637,903	19.2
Kansas	5,439,948	1,445,000	1,644,652	489,000	2,200,000	650,000	11,868,600	41.3
Kentucky	6,424,666	432,817	2,646,979	N/A	867,063	N/A	10,371,525	31.3
Louisiana	4,781,469	N/A	5,958,309	327,863	N/A	271,759	11,339,400	−11.5
Maine	4,702,828	1,075,174	1,532,942	N/A	N/A	N/A	7,310,944	−15.3
Maryland	26,174,940	0	4,172,301	1,040,082	1,160,671	9,817,619	42,365,613	50.5
Massachusetts	39,510,423	N/A	9,400,000	N/A	N/A	N/A	48,910,423	36.1
Michigan	29,057,429	N/A	11,784,533	3,146,094	8,119,634	28,750,167	80,857,857	23.4
Minnesota	2,279,758	N/A	3,099,054	N/A	N/A	N/A	5,378,812	7.4
Mississippi	2,449,962	0	1,047,511	2,029,886	N/A	N/A	5,527,359	−19.0
Missouri	10,200,885	N/A	5,117,343	790,412	266,384	N/A	16,375,024	43.6
Montana	503,643	1,984,506	971,190	840,832	1,572,653	3,990,532	9,863,356	22.4
Nebraska	4,672,559	0	1,540,230	0	567,118	838,602	7,618,509	23.2
Nevada	2,338,443	0	1,241,056	0	157,161	2,263,171	[3] 5,999,831	−8.4
New Hampshire	1,376,037	N/A	1,015,121	N/A	N/A	15,075	2,406,233	3.0
New Jersey	21,985,000	0	10,171,000	987,000	2,015,000	3,500,000	38,658,000	73.3
New Mexico	2,461,248	707,135	1,706,508	0	0	9,065,277	[1] 13,940,168	2.7
New York	190,213,527	4,657,900	35,874,500	8,331,900	33,429,300	178,015,215	[1,4,5] 450,522,342	45.6
North Carolina	12,860,884	N/A	3,933,569	322,977	17,733,189	N/A	34,850,619	19.4
North Dakota	1,235,977	0	1,559,620	0	0	0	2,795,597	57.3
Ohio	16,603,294	3,251,365	8,558,398	2,009,863	1,491,494	4,847,535	36,761,949	2.2
Oklahoma	4,510,066	4,988,744	1,974,736	108,270	N/A	N/A	11,581,816	95.5
Oregon	7,217,339	20,345,307	3,461,952	1,476,700	5,256,582	2,413,698	40,171,578	268.0
Pennsylvania	30,475,690	6,746,377	12,081,562	154,000	6,261,976	18,493,976	74,213,581	12.9
Puerto Rico	14,601,022	2,384,789	3,969,492	467,994	0	0	21,423,297	22.4
Rhode Island	7,407,973	N/A	1,924,373	N/A	N/A	N/A	[6,7] 9,332,346	28.0
South Carolina	8,224,370	4,167,536	2,479,164	1,322,780	4.180,000	3,702,138	24,075,988	92.4
South Dakota	422,763	514,158	956,272	51,938	658,323	1,373,429	[6] 3,976,883	−1.0
Tennessee	6,480,412	1,620,366	3,176,803	1,072,414	3,391,608	4,110,775	19,852,378	96.5
Texas	4,837,251	N/A	9,001,730	688,495	N/A	N/A	[8] 14,527,476	−28.9
Utah	6,105,571	1,172,441	2,099,714	760,830	2,592,114	4,386,968	17,117,638	32.4
Vermont	2,751,140	167,000	1,114,620	0	0	1,408,703	5,441,463	44.0
Virginia	14,295,104	N/A	4,248,498	693,176	7,695,950	4,606,291	31,539,019	16.7
Washington	19,713,486	N/A	4,249,712	748,403	N/A	N/A	24,711,601	14.1
West Virginia	2,636,497	1,169,522	1,422,697	226,881	115,558	2,229,348	7,800,503	4.7
Wisconsin	37,702,567	7,114,253	4,498,443	8,570,000	2,921,724	5,164,125	65,971,112	25.1
Wyoming	2,742,561	0	467,487	0	252,598	N/A	3,462,646	−10.8
Totals	819,807,824	104,310,843	272,570,630	51,766,318	164,842,427	396,450,971	1,809,749,013	31.2
Percent of total	45.3	5.8	15.1	2.9	9.1	21.9	100.0	

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[1] Figures represent allocated funds rather than expenditures.

[2] Alcohol/Drug Abuse Block Grant Includes $324,272 Alcohol and Drug Treatment and Rehabilitation (ADTR) Block Grant monies.

[3] County or local agencies category includes required matching funds.

[4] Other sources category includes Medicaid, client fees, and Juvenile Justice Prevention Funds.

[5] Other State agency category includes lab revenues, methadone registry, capital construction, Medicaid MIS, and suballocations from the Department of Social Services.

[6] Figures represent an estimate of expenditures.

[7] State Alcohol and Drug Agency category includes substance abuse detoxification facility, DUI program, and TASC.

[8] Other Federal category includes ADTR Block Grant Funds.

N/A = information not available.

SOURCE: State Alcohol and Drug Abuse Profile, fiscal year 1987; data are included for "only those programs which received at least some funds administered by the State Alcohol-Drug Agency during the State's fiscal year 1987."

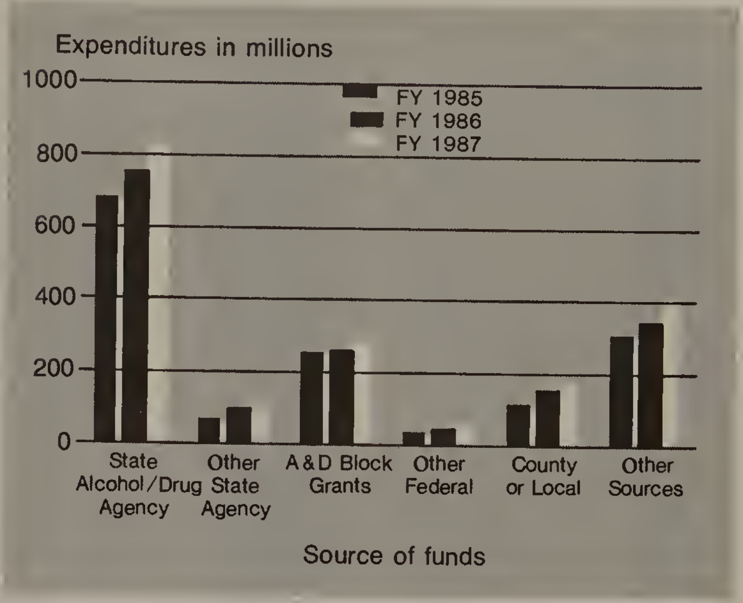
As illustrated in figure 1, this total includes $819.8 million (45.3 percent) from State alcohol and drug agency sources, $104.3 million (5.8 percent) from other State agency sources, $272.6 million (15.1 percent) from Alcohol, Drug Abuse, and Mental Health Services Block Grant, $51.8 million (2.9 percent) from other Federal Government sources, $164.8 million (9.1 percent) from county or local agency sources, and $396.5 million (21.9 percent) from other sources (for example, reimbursements from private health insurance, client fees, and court fines or assessments for treatments imposed on intoxicated drivers).

Since the data include information only on those programs "which received at least some funds administered by the State Alcohol/Drug Agency during the State's Fiscal Year 1987," caution needs to be exercised in using and interpreting these data. In some States, complete information is not available on all funding sources, even for State alcohol and drug agency-supported programs. Consequently, the percentages presented in table 1 should be used only as gross estimates of the overall level of funding from various sources. It is likely that the "Other State," "Other Federal," "County or local," and particularly, "Other sources" categories actually contributed more funds than the figures indicate.

The specific levels of fiscal support from the different sources vary considerably among the States. However, for all States combined and for most States individually, the single largest source of funding during fiscal year 1987 for alcohol and drug services was State revenues. In 38 States and Puerto Rico, the State alcohol and drug agency funds constituted the single largest source of funding. The largest revenue source in five States and Guam was the Federal Government, primarily through the alcohol and drug abuse share of the Alcohol, Drug Abuse, and Mental Health Services Block Grant, but also partially through the new Part C, Emergency Alcohol and Drug Treatment and Rehabilitation Block Grant. In three States and the District of Columbia, other State agency funds were the major sources of support; in another three States, other sources constituted the largest funding source; and within one State, North Carolina, county or local agencies were the largest source of fiscal support for alcohol and drug abuse services.

A comparison of total expenditures from all sources for alcohol and drug abuse services for fiscal years 1985 to 1987 in the 53 State agencies reporting data for all 3 years reflects a total dollar increase of $430,266,246, or a 31.2 percent increase (table 1) (fig. 2). Although this increase reflects a positive growth in the overall national fiscal environment, eight State agencies reported a decrease in total expenditures from fiscal year 1985 to 1987. Also, in a few States, the increase in expenditures may reflect not a true increase in services but an improvement in the reporting or data collection system. In addition, the dollar increase from fiscal year 1985 to 1986 was 19.0 percent, while the increase from fiscal year 1986 to 1987 was only 10.2 percent.

Figure 2. Comparison of expenditures for State-supported alcohol and drug abuse services by funding source for fiscal years 1985, 1986, and 1987



NOTE: Some of the apparent increases in expenditures may be related to an improvement in the State's ability to collect and provide data from different funding sources. The "Other Sources" category includes funding from sources such as client fees, court fines, and reimbursements from private health insurance.

SOURCE: State Alcohol and Drug Abuse Profile, FY 1987; data are included for "only those programs which received at least some funds administered by the State Alcohol/Drug Agency during the State's Fiscal Year 1987."

Expenditures by program activity. Expenditures during fiscal year 1987 are presented by State for three program activities—treatment, prevention, and other (table 2).

As noted previously, the total expenditures for the 50 States, the District of Columbia, Guam, and Puerto Rico during fiscal year 1987 in those programs that received at least some State alcohol and drug agency funds were $1.8 billion. Of the total, nearly $1.4 billion (76.5 percent) was expended for treatment, $224.2 million (12.6 percent) for prevention, and $194.4 million (10.9 percent) for other activities (for example, training, research, and administration). Only one State was unable to report the breakout of $31.5 million (or 1.7 percent of the total) by the type of program activity.

Table 2. Expenditures for State supported alcohol and drug abuse services by State and by type of program activity for fiscal year 1987

State	Type of program activity			
	Treatment	Prevention	Other	Total
Alabama	6,089,157	751,365	462,578	7,303,100
Alaska	13,721,500	5,842,500	1,964,100	21,528,100
Arizona	20,088,213	1,596,622	692,802	[2] 22,377,637
Arkansas	5,627,807	596,203	430,917	6,654,927
California	156,032,000	32,839,000	32,201,000	[2] 221,072,000
Colorado	20,395,555	3,486,440	1,564,223	25,446,218
Connecticut	33,082,662	3,040,958	4,342,595	40,466,215
Delaware	2,989,029	231,956	785,745	4,006,730
District of Columbia	20,811,317	1,129,253	4,638,615	26,579,185
Florida	68,293,843	6,319,401	168,477	74,781,721
Georgia	30,391,265	785,022	969,587	32,145,874
Guam	85,024	108,086	58,783	251,893
Hawaii	3,450,156	511,017	388,085	4,349,258
Idaho	2,079,500	135,500	190,900	2,405,900
Illinois	54,150,800	5,358,400	6,000,700	65,509,900
Indiana	19,017,428	1,495,986	797,552	21,310,966
Iowa	12,028,466	2,609,437	0	14,637,903
Kansas	8,998,914	1,806,216	1,063,470	11,868,600
Kentucky	8,644,457	1,086,914	640,154	10,371,525
Louisiana	8,543,168	1,641,121	1,155,111	11,339,400
Maine	5,999,542	783,067	528,335	7,310,944
Maryland	38,306,552	1,062,530	2,996,531	42,365,613
Massachusetts	37,030,181	6,179,393	6,700,849	48,910,423
Michigan	58,895,864	13,554,896	8,407,097	80,857,857
Minnesota	3,399,024	1,025,783	954,005	5,378,812
Mississippi	4,274,317	223,042	1,030,000	5,527,359
Missouri	14,524,828	812,945	1,037,251	16,375,024
Montana	8,847,686	700,947	314,723	9,863,356
Nebraska	6,455,183	846,640	316,686	7,618,509
Nevada	5,094,070	299,715	606,046	5,999,831
New Hampshire	1,430,691	580,955	394,587	2,406,233
New Jersey	28,911,000	7,002,000	2,745,000	38,658,000
New Mexico	12,899,675	968,548	71,945	[2] 13,940,168
New York	356,563,836	58,912,506	35,046,000	[2] 450,522,342
North Carolina	2,675,117	862,507	31,312,995	34,850,619
North Dakota	2,551,087	97,113	147,397	2,795,597
Ohio	26,640,460	4,595,901	5,525,588	36,761,949
Oklahoma	10,701,915	469,136	410,765	11,581,816
Oregon	27,511,392	11,147,811	1,512,375	40,171,578
Pennsylvania	54,801,483	12,450,078	6,962,020	74,213,581
Puerto Rico	13,781,163	2,245,910	5,396,224	21,423,297
Rhode Island	7,980,013	772,175	580,158	9,332,346
South Carolina	15,532,386	6,975,115	1,568,487	24,075,988
South Dakota	3,251,871	338,744	386,268	3,976,883
Tennessee	12,585,331	4,414,815	2,852,232	19,852,378
Texas	8,250,992	3,340,949	2,935,535	14,527,476
Utah	11,167,699	5,217,395	732,544	17,117,638
Vermont	3,889,139	871,164	681,160	5,441,463
Virginia	N/A	N/A	N/A	[3] 31,539,019
Washington	22,676,269	865,000	1,170,332	24,711,601
West Virginia	6,386,672	1,067,256	346,575	7,800,503
Wisconsin	49,509,626	4,492,257	11,969,229	65,971,112
Wyoming	2,546,660	648,597	267,389	3,462,646
Totals	1,359,591,985	224,196,287	194,421,722	1,809,749,013
Percent of total	76.5	12.6	10.9	. . .

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[1] Other category includes other activities beyond treatment or prevention services (for example, training, research, and administration).

[2] Figures represent allocated funds rather than expenditures.

[3] State was unable to differentiate among program categories.

NOTE: N/A = information not available.

SOURCE: State Alcohol and Drug Abuse profile, fiscal year 1987; data are included for "only those programs which received at least some funds administered by the State Alcohol/Drug Agency during the State's Fiscal Year 1987."

Table 3. Number of alcohol and drug treatment units which received funds administered by the State alcohol and drug agency for fiscal year

State	Treatment units				Estimated percent of total treatment units funded by State agency
	Alcohol	Drug	Combined alcohol and drug	Total	
Alabama	13	5	16	[1] 46	51
Alaska	0	2	45	47	82
Arizona	38	18	122	178	67
Arkansas	5	4	19	28	61
California	635	249	N/A	884	60
Colorado	63	7	23	93	34
Connecticut	31	27	42	100	64
Delaware	5	2	6	13	50
District of Columbia	4	9	2	15	60
Florida	55	76	42	173	80
Georgia	0	0	43	43	N/A
Guam	0	0	1	1	100
Hawaii	3	2	17	22	80
Idaho	0	0	20	20	64
Illinois	318	93	26	437	45
Indiana	0	0	42	42	20
Iowa	0	0	29	29	53
Kansas	0	0	65	65	40
Kentucky	0	0	132	132	40
Louisiana	0	0	43	43	33
Maine	0	0	34	34	58
Maryland	62	42	20	124	51
Massachusetts	33	11	254	298	N/A
Michigan	13	4	248	265	49
Minnesota	1	2	43	46	17
Mississippi	50	1	22	73	75
Missouri	3	5	80	88	42
Montana	1	2	27	30	75
Nebraska	0	0	127	127	78
Nevada	2	1	25	28	40
New Hampshire	6	2	17	25	36
New Jersey	121	50	17	188	[2]
New Mexico	22	11	20	53	N/A
New York	300	513	27	840	[3]
North Carolina	23	1	41	65	73
North Dakota	0	0	7	7	25
Ohio	92	47	42	181	[4]45
Oklahoma	0	0	48	48	50
Oregon	52	9	39	100	53
Pennsylvania	40	26	459	525	68
Puerto Rico	8	160	5	[5] 173	100
Rhode Island	19	7	4	30	70
South Carolina	2	0	38	40	60
South Dakota	0	0	18	18	64
Tennessee	1	2	51	54	60
Texas	0	3	72	75	26
Utah	5	0	65	70	77
Vermont	0	0	27	27	90
Virginia	20	6	64	90	75
Washington	37	27	60	124	44
West Virginia	0	0	29	29	85
Wisconsin	0	2	322	324	80
Wyoming	0	0	22	22	85
Totals	2,083	1,428	3,109	6,632	. . .
Percent of total	31.4	21.5	46.9	100.0	. . .

[1] Includes 12 prevention units.
[2] 50 percent of alcohol units, 90 percent of drug units, New Jersey.
[3] 81 percent of alcohol units, 65 percent of drug units, New York.
[4] Alcohol units only.
[5] Total includes 91 private day treatment units.

NOTE: N/A = information not available.

SOURCE: State Alcohol and Drug Abuse Profile, fiscal year 1987; data are included for only those programs "which received funds administered by the State Alcohol/Drug Agency during Fiscal Year 1987."

Table 4. Number of alcohol client treatment admissions, by sex and State for fiscal year 1987

State	Male	Female	Not reported	Total
Alabama	3,922	1,482	0	5,404
Alaska	6,712	2,239	0	8,951
Arizona	15,700	4,930	0	20,630
Arkansas	6,741	1,097	0	7,838
California	100,900	23,300	0	[1] 124,200
Colorado	35,001	6,226	10,890	52,117
Connecticut	10,931	2,679	0	13,610
Delaware	3,449	612	4	4,065
District of Columbia	4,482	1,121	0	5,603
Florida	48,613	14,048	0	62,661
Georgia	33,521	7,659	0	41,180
Guam	N/A	N/A	40	40
Hawaii	776	280	0	1,056
Idaho	3,848	1,089	0	4,937
Illinois	55,470	13,138	532	69,140
Indiana	9,962	3,079	0	13,041
Iowa	14,574	3,834	0	18,408
Kansas	8,885	1,681	2	10,568
Kentucky	11,036	2,272	0	13,308
Louisiana	5,136	1,474	0	6,610
Maine	14,108	4,756	0	[2] 18,864
Maryland	16,701	3,459	0	20,160
Massachusetts	71,108	15,748	0	86,856
Michigan	29,660	7,599	218	37,477
Minnesota	36,765	6,647	447	43,859
Mississippi	6,121	925	88	7,134
Missouri	18,266	3,447	0	21,713
Montana	5,493	3,927	0	9,420
Nebraska	14,649	4,639	0	19,288
Nevada	2,423	578	7,495	10,496
New Hampshire	1,969	791	46	2,806
New Jersey	20,263	5,418	0	25,681
New Mexico	9,316	2,188	14	11,518
New York	109,916	40,654	0	[3] 150,570
North Carolina	16,442	2,917	0	19,359
North Dakota	829	322	0	1,151
Ohio	14,412	4,353	0	18,765
Oklahoma	5,733	1,465	1,252	8,450
Oregon	23,532	7,116	0	30,648
Pennsylvania	31,712	7,840	0	39,552
Puerto Rico	3,068	161	0	3,229
Rhode Island	5,072	1,120	0	6,192
South Carolina	19,709	4,011	930	24,650
South Dakota	3,389	1,480	0	4,869
Tennessee	6,373	1,862	0	8,235
Texas	4,982	1,557	19,436	25,975
Utah	9,942	1,624	0	11,566
Vermont	2,624	1,024	0	3,648
Virginia	36,392	6,322	0	42,714
Washington	30,710	7,774	0	38,484
West Virginia	7,606	1,620	0	9,226
Wisconsin	39,946	13,077	11,493	[4] 64,516
Wyoming	5,062	1,973	0	[5]7,035
Totals	1,003,952	260,634	52,887	1,317,473
Percent of total	76.2	19.8	4.0	100.0

[1] Alcohol client admissions data are estimated.

[2] Includes both alcohol and drug admissions; approximately 76 percent of total admissions are alcohol and 24 percent are drug.

[3] All client admissions data are for calendar year 1986 and are estimated.

[4] Not reported column represents duplicate admissions.

[5] Includes both alcohol and drug admissions.

N/A = Information not available.

SOURCE: State Alcohol and Drug Abuse Profile, fiscal year 1987; data are included for only those programs "which received some funds administered by the State Alcohol Agency during fiscal year 1987."

Over the past several years, many States have substantially increased their commitment to and financial expenditures for prevention programs. However, within every State agency except Guam's, the expenditures for treatment remain much higher than those for prevention. Overall, the expenditures for treatment are more than six times that for prevention.

Characteristics of treatment units. A total of 6,632 alcohol and drug treatment units received funds administered by the State alcohol and drug agencies in fiscal year 1987. With regard to their orientation, 2,083 (31.4 percent) were identified as alcohol units, 1,428 (21.5 percent) as drug units, and 3,109 (46.9 percent) as combined alcohol and drug treatment units (table 3).

An estimate of the percentage of alcohol and drug treatment units in the State that received State alcohol and drug agency-administered funds in fiscal year 1987 was provided by 47 States, the District of Columbia, Guam, and Puerto Rico. The estimate ranged from a low of 17 percent in Minnesota to a high of 100 percent in Guam and Puerto Rico (table 3).

Client Admissions to Treatment Services

Each State alcohol and drug agency was asked to provide information on client admissions to the treatment units that received State agency funds during fiscal year 1987. However, treatment units that received only partial funding from the State agency were instructed to report data on all client admissions supported by State agency funds.

All but four States have combined alcohol and other drug abuse treatment responsibilities within one agency. A number of these agencies have established combined treatment systems (for example, substance abuse, chemical dependency) or client reporting systems and preferred to report combined data on alcohol and drug clients. However, in response to a specific request from NIAAA and NIDA (each agency has a distinct mandate), NASADAD asked the States separate questions relating to alcohol and other drug abuse treatment services so that data collected would be generally consistent with previously collected data.

Information on client admissions to treatment services is organized in two sections: client admissions by environment and type of care and a comparison of treatment admissions for alcohol and for other drugs. Data are also presented by clients' sex, age, and race-ethnicity.

Client admissions by environment and type of care. Each State alcohol (and combined alcohol and drug) agency was asked to provide data on the "number of ALCOHOL treatment client admissions during fiscal year 1987." The information requested included admissions data organized by environment (hospital or nonhospital) and by type of care (detoxification, rehabilitation-residential, or outpatient) (data not shown).

All 50 State agencies, the District of Columbia, Guam, and Puerto Rico provided at least some data on the number of total alcohol client treatment admissions during fiscal year 1987. The total of reported admissions for alcohol treatment was 1,317,473. Of these admissions, approximately 84.6 percent (1,114,334 admissions) were to nonhospital units. However, three States that reported admissions to nonhospital units did not have data on the admissions to hospital units; therefore, the actual number and percentage of the hospital admissions are likely to be higher than indicated. Forty-six States, the District of Columbia, Guam, and Puerto Rico reported a total of 142,777 client admissions to hospital-based treatment units.

Most States also reported data on alcohol client treatment admissions by type of care (detoxification, rehabilitation-residential, or outpatient) as well as by environment (hospital or nonhospital) (data not shown). Hospitals were used by 16.8 percent of those clients who required detoxification services and 17.1 percent of those clients who received rehabilitation-residential services. However, the proportion of hospital to nonhospital admissions is somewhat lower for those clients who required outpatient services. With regard to outpatient services, hospital facilities were used for only 5.1 percent of the client admissions, while 94.9 percent of outpatient services were delivered in a nonhospital setting. Since some State agencies reported data in some categories but not in others, caution should be exercised in the interpretation and use of the percentages noted previously.

Client admissions data by sex, age, and race-ethnicity. Data on clients by sex, age, and race-ethnicity categories were supplied by the State agencies. All 50 States and the District of Columbia reported data by sex (table 4).

Approximately 76.2 percent of the alcohol client admissions were male, and 19.8 percent were female. Data on sex were not reported for 4.0 percent of the alcohol client admissions.

Forty-eight States, Guam, and the District of

Columbia were able to report data for all or at least some age categories requested (data not shown). The percent of client admissions within each of the age-range categories requested follows:

Age group (years)	*Percent of admissions*
Under 18	4.1
18–20	4.1
21–24	10.7
25–34	27.4
35–44	21.7
45–54	9.7
55–64	5.3
65 and over	1.8
Not reported	15.3

A total of 42 State agencies and the District of Columbia reported at least some relevant data by age-sex subgroups (data not shown). A number of States have established different age group categories and therefore were unable to retrieve or report client information according to some or all of the requested categories. The data indicate a male-female mix of 66 percent to 34 percent in the youngest age group (under 18), increasing to 84 percent male in the oldest group (65 and over).

With regard to admissions information by race-ethnicity, a total of 49 State agencies, the District of Columbia, Guam, and Puerto Rico provided at least partial data (data not shown). Among the States reporting data, the percentage of client admissions by race-ethnicity categories specified were as follows:

Race-ethnicity	*Percent of admissions*
White, not of Hispanic origin	69.7
Black, not of Hispanic origin	15.6
Hispanic	5.5
Asian or Pacific Islander	0.2
Native American	3.6
Other	0.3
Not reported	5.2

Since some State agencies reported data in some categories but not in others, caution should be exercised in the interpretation and use of the percentages for both age and race-ethnicity.

Alcohol and Drug Treatment Clients Compared

The following data demonstrate the magnitude of alcohol problems compared with other drug problems, as well as similarities and differences in client demographics:

- Total alcohol and other drug client admissions to treatment: total alcohol admissions, 1,317,473; total other drug admissions, 450,553.
- Admissions by sex: alcohol admissions, 76.2 percent male, 19.8 percent female, and 4.0 percent not reported; drug admissions, 61.3 percent male, 31.2 percent female, and 7.5 percent not reported.
- Admissions by age: alcohol admissions, 4.1 percent under age 18 and 27.4 percent ages 25–34; drug admissions, 14.3 percent under age 18 and 33.5 percent ages 25–34.
- Admissions by race-ethnicity: alcohol admissions, 69.7 percent white, 15.6 percent black, 5.5 percent Hispanic; drug admissions, 48.3 percent white, 20.7 percent black, 9.8 percent Hispanic.
- Admissions by type of drug: alcohol, 1,317,473; all other drugs combined, 450,553; including heroin 98,489, cocaine 84,707, marijuana-hashish 83,740.

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The Indian Health Service Approach to Alcoholism Among American Indians and Alaska Natives

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Synopsis ....

The transfer to the Indian Health Service (IHS) of 158 alcohol treatment programs that had been administered by the National Institute on Alcohol Abuse and Alcoholism began in 1978. Today, approximately 300 alcohol and substance abuse treatment programs offer services to American Indians, among them primary residential treatment, halfway houses, outreach, and aftercare. This system provides a national network upon which additional activities may be established.

Along with increasing its attention to health promotion and disease prevention, the IHS has moved toward the prevention of alcoholism. A variety of preventive programs are in place that emphasize improved self-image, value and attitude clarification, decision-making, and physical and emotional effects of alcohol and substance abuse. Many begin as Head Start programs and continue through adulthood.

In 1986, after consulting with both academic and tribal experts, the IHS devised a strategic plan for alcoholism control that stresses comprehensive care and prevention activities; it serves as a guide for further program development. The Secretary of Health and Human Services created a Task Force on Indian Alcoholism in 1986 to serve as a coordinating body for activities carried out by the IHS and other agencies and units of the Department. Passage of the Anti-Drug Abuse Act in 1986 added resources for the development of adolescent treatment centers and, more importantly, for community-based pre- and post-residential care for youths and their families. Concomitant with these initiatives have been several instances of increased attention by various tribes to the problem of alcoholism.

The IHS strategic plan, together with the Secretary's initiative, the Anti-Drug Act, and tribal actions, has added substantial momentum to efforts directed at controlling alcoholism among American Indians. Although the mortality rate from alcoholism is about four times greater for the American Indian population than for the entire U.S. population, it decreased from 54.5 per 100,000 population to 26.1 between 1978 and 1985—a reduction of 52 percent.

The philosophy of the IHS in emphasizing prevention of disease and promotion of wellness provides an opportunity for continuing the considerable progress already made. The critical and decisive role played by the Indian communities themselves will determine whether ultimate success can be achieved.

ALCOHOLISM AMONG AMERICAN INDIANS, although known to be a serious problem, was not the primary concern among physicians and health care workers for several years after the Indian Health Service (IHS) was established in 1955. Until the late 1960s, the major focus was on controlling acute and chronic infectious diseases that were both devastating and more amenable to therapy.

In 1969, when success in controlling infectious diseases had been realized, and the effects of alcoholism had become increasingly devastating, the IHS declared the intensification of efforts to

Table 1. Leading causes of death among American Indians and Alaska Natives, all ages, in States with reservations, 1983–85 (mortality rates per 100,000 population)

Causes	Number of deaths[1]	Mortality rate[2]
All causes	20,110	474.0
Diseases of the heart	4,685	110.4
Accidents	3,218	75.9
Motor vehicle	1,753	41.3
Other accidents	1,465	34.5
Malignant neoplasms	2,725	64.2
Cerebrovascular diseases	969	22.8
Chronic liver diseases and cirrhosis	924	21.8
Pneumonia and influenza	682	16.1
Diabetes mellitus	676	15.9
Homicide	597	14.1
Suicide	581	13.7
Certain conditions originating in the perinatal period	387	9.1
All other	4,666	...

[1] The number of deaths in a 3-year period is used because of the small numbers of deaths each year.

[2] Because the denominator is a 3-year sum of the population, the rate may be regarded as an annual average rate.

NOTE: Connecticut, Rhode Island, and Texas were included as reservation States beginning in 1983, and Alabama in 1984. Data include 17 deaths with age not reported.

deal with alcoholism to be a high priority. The Comprehensive Alcohol Abuse Treatment, Prevention, Rehabilitation Act of 1970 (PL 91-616) authorized that treatment facilities be established for Indian people. Initially, the responsibility for carrying out the programs was placed with the National Institute on Alcohol Abuse and Alcoholism (NIAAA).

The transfer of ''mature'' programs from NIAAA to IHS was begun in 1978, when 36 programs were transferred. By 1983, after having received support from NIAAA for 6 years, a total of 158 programs had been transferred. In developing these programs, IHS focused on (*a*) defining the scope of alcohol treatment and prevention at all levels, (*b*) improving the quality of care provided to persons in alcohol treatment programs, and (*c*) seeking ways to expand services available to Indian people.

Mortality Rates

Although alcoholism as a specific diagnosis (coded 303 or 305.0 in the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM)) does not occur as one of the leading causes of death among American Indians, its impact upon the mortality of Indians may be inferred from data shown in table 1, which depicts the 10 leading causes of death. Because the majority of deaths classed as ''accidents'' seem to be alcohol-related, and most of the deaths from chronic liver disease are, in fact, from cirrhosis that is associated with alcoholism, it is clear that alcoholism and its secondary effects play a preponderant role in the overall mortality of American Indians.

The mortality rate, expressed as rate per 100,000 population, for chronic liver disease and cirrhosis nearly equals that for cerebrovascular disease and exceeds that for diabetes, or pneumonia and influenza. At 29.2 per 100,000, the rate is three times greater than that for the general U.S. population. For Indians ages 15 to 24, it is the ninth leading cause of death; for Indians ages 25 to 44, it is the second leading cause of death, with a rate of 31.6 per 100,000 compared with 5.9 per 100,000 for the general population in that age group (*1*).

Age-adjusted deaths and mortality rates for Indians compared with the category ''U.S. all races'' for the 8-year period 1978–85 are shown in table 2. The mortality rate for Indians decreased from 54.5 per 100,000 in 1978 to 26.1 per 100,000 in 1985, a reduction of 52 percent. The comparable figures for the category U.S. all races were 8.1 per 100,000 in 1978 and 6.2 per 100,000 in 1985, a decrease of 23 percent.

In table 3 are shown the deaths and death rates for all alcohol-related deaths in each IHS Area for the 3-year period 1981–83 (*2*). The wide variation in population precludes a comparison of the numbers of deaths among the various Areas. Areas with the highest mortality rates from alcoholism are Billings, MT (136.9 per 100,000) and Phoenix, AZ (103.6 per 100,000). These rates compare with the rates 32.7 per 100,000 and 34.1 per 100,000, respectively, for the Alaska and Navajo Areas. Data for various rates, including mortality, in California and Oklahoma, where discrepancies appear—possibly as the result of persons being identified as Indians at the time of birth but not at death—make it very difficult to reach accurate conclusions. In any case, the mortality rate in each Area is considerably greater than that for the U.S all races category.

IHS's Strategic Plan

In 1985, the Director, IHS, convened a group of persons who have a wide range of skills and experience to review existing alcoholism programs and to make recommendations regarding the future direction for IHS. A series of working groups

Table 2. Deaths from alcoholism[1], age-adjusted mortality rates[2], and ratios of age-adjusted mortality rates, Indians to U.S. all races, for American Indians and Alaska Natives in States with reservations, 1978–85

Calendar year	Number of deaths		Age-adjusted rates		Ratio: Indian to U.S. all races
	Indian and Alaska Native	U.S. all races	Indian and Alaska Native	U.S. all races	
1985	281	15,844	26.1	6.2	4.2
1984	316	15,706	30.0	6.2	4.8
1983	293	15,424	28.9	6.1	4.7
1982	298	15,596	30.7	6.4	4.8
1981	338	16,745	35.8	7.0	5.2
1980	382	17,742	41.3	7.5	5.5
1979	398	17,064	45.1	7.4	6.1
1978	437	18,490	54.5	8.1	6.7

[1] Deaths from alcoholic psychoses, alcohol dependence syndrome, and chronic liver disease and cirrhosis specified as alcoholic.

[2] Mortality rates per 100,000 population.

made more than 100 recommendations covering the entire field of alcohol abuse. A major theme of the review was that future efforts should be prevention oriented, especially prevention targeted to youth. The discussions and recommendations became the basis for a strategic plan (3) dealing with alcohol abuse as a major policy issue, resource management, quality assurance, coordination of activities, training and research. Fifty specific action steps with time frames for accomplishment are contained in the plan. This plan has served as an important guide for the IHS in its continued efforts to control alcoholism among Indian people.

Table 3. Alcohol abuse mortality[1]

Population	Deaths	
	Number	Rate
United States, 1982	31,993	12.3
IHS Areas, 1981–83:		
All areas	1,025	52.7
Aberdeen	110	89.3
Alaska	46	32.7
Albuquerque	68	75.0
Bemidji	35	39.7
Billings	102	136.9
California	33	20.9
Nashville	23	35.0
Navajo	107	34.1
Oklahoma	149	32.8
Phoenix	183	103.6
Portland	136	79.0
Tucson	33	97.9

[1] National Center for Health Statistics data for underlying cause of death. Included are deaths from alcohol dependence syndrome (ICD–9 code 303), alcoholic psychoses (ICD–9 code 291), nondependent use of drugs (ICD–9 code 305), and chronic liver disease and cirrhosis (ICD–9 code 571.0–571.9).

[2] Because of the small numbers, calculations are made on 3-year totals, then expressed as an annual age-adjusted rate per 100,000 population.

Community and School Prevention Activities

For several years, the IHS and Indian communities have been increasing the number of alcoholism prevention activities they offer. Adults who are served by IHS-funded programs have been provided routinely with education about alcohol and its effects. Expectant mothers receiving prenatal care at IHS clinics and hospitals are supplied information about fetal alcohol syndrome. A number of Indian communities have developed an impressive array of prevention services and resources.

Owan and coworkers (*4*) conducted a survey of schools and community prevention activities among Indian communities during the period October 1986 to March 1987. They collected information from 420 schools and 160 tribal groups in an effort to determine the extent of prevention programs aimed specifically at the youth. They found that a number of schools used curriculums such as ''Here's Looking at You,'' ''Project Charlie,'' and ''Beginning Alcohol and Addiction Basic Education Studies (BABES)'' (*see box*, page 625), which emphasize values and attitude clarification, decision-making, and the physical and emotional effects of alcohol and substance abuse. More than 1,500 persons were involved in prevention programs in schools attended by Indian children, including teachers and their aides, Indian parents, and alcoholism counselors, among others. One of the benefits of these programs is that they prompted a number of referrals of students for counseling. In addition, several student and parent groups were formed, including peer counseling groups, Students Against Drunk Driving, Chemical People, and Mothers Against Drunk Driving.

The study showed that community-based services were provided for men and women, adolescents, parents, and single parents. Many of the school-based curriculums were also used in community-based programs. Special activities such as parent effectiveness training, ‘‘Children Are People,’’ ‘‘Trails,’’ and ‘‘Circles of Life’’ (see box) were offered. A total of 312 communities and 31,946 people participated in the programs.

Community-based programs provided alcohol and drug education and activities designed to build self-esteem and coping skills, develop decision-making skills, promote family bonding and enrichment, and teach effective parenting. The survey revealed a wealth of activities and information about the programs, including the information that local Indian people and substance abuse program directors were appearing on local public service TV stations and making radio presentations to discuss substance abuse issues of concern to their communities.

Fetal Alcohol Syndrome

After a major study in 1983 of the incidence and prevalence of fetal alcohol syndrome (FAS) among selected tribes, the IHS initiated a training program for health care workers in every IHS Service Unit. A total of 1,279 community-based workers were trained to promote understanding of FAS and fetal alcohol effect (*4*) and to assist communities in the development of prevention and intervention programs. The IHS funds research directed at defining the extent of FAS and long-term followup of children with FAS. Attention to the FAS-associated problems continues to be an integral part of prevention activities and is especially emphasized in prenatal clinics.

Alcohol-Related Research

Participants of the Alcoholism Program Review of 1985 made four major recommendations for alcoholism and substance abuse research (*4*):

- the focus of IHS-funded research should be prevention,
- applied research should be emphasized,
- studies on the characteristics of American Indians and Alaska Natives who do not abuse substances should be encouraged,
- IHS should update morbidity-mortality data at the service unit level.

The implementation of these recommendations resulted in the development of model demonstration programs for health promotion-disease prevention that focus on alcohol or other drugs. Morbidity data may still be underreported, particularly in areas such as California and Oklahoma, as mentioned earlier, and attention is being given to determining the extent and effects of this problem. Concerted training efforts for physicians and other health care providers are expected to contribute to more accurate reporting of alcohol- and other drug-related morbidity and mortality. Finally, the study of various health needs of American Indian and Alaska Native people remains a high priority. The IHS and the Alcohol, Drug Abuse, and Mental Health Administration are jointly engaged in an effort to stimulate research concerning alcoholism among Indians. An important part of this effort is the identification and support of young Native American researchers who may be interested in pursuing a research career in this field.

Secretary’s Initiative

As part of his special emphasis programs, the Secretary of Health and Human Services, Otis R. Bowen, MD, established, on June 3, 1986, the Secretary’s Indian Alcoholism Initiative, with the IHS having the lead responsibility. The charge to the task group established to carry out the initiative was

1. to ensure a permanent, coordinated effort to combat the serious health problem of alcohol and substance abuse among American Indians and Alaska Natives by drawing on the Department’s expertise in health promotion and disease prevention,
2. to restructure activities permanently, rather than provide a time-limited campaign,
3. to provide training of health professionals,
4. to involve lay efforts within tribes, including coordination with the Indian schools.

As part of this effort, the Secretary created an interagency task force made up of representatives from the Administration on Aging; Alcohol, Drug Abuse and Mental Health Administration; Administration for Children, Youth, and Families; and the Office of Minority Health. This initiative has resulted in several new activities being carried out through memorandums of agreement between the IHS and other agencies. For example, the Administration on Aging provides 11 grants dealing with Indian alcoholism. The Administration for Native

Americans provides five grants for resolving chemical dependency and four for youth entrepreneurial activities. The Administration for Children, Youth, and Families is studying placement issues with families involved with alcohol abuse, in addition to addressing alcohol and drug abuse in the home through 104 Head Start programs.

Twelve grants have been awarded by the Alcohol, Drug Abuse, and Mental Health Administration on issues dealing with the impact of mental illness on prevention, treatment, and rehabilitation of alcohol abusing patients, as well as basic and applied research in preventive intervention. The Office of Minority Health has created a task force to disseminate and utilize information on minority projects in order to identify and assess successful projects.

A second major effort resulting from the Secretary's Initiative is the development of a task force composed of representatives of the Administrations within the Department, the IHS, and the Bureau of Indian Affairs (BIA). This task force monitors activities concerning the initiative and reports to the Secretary on progress.

1986 Anti-Drug Abuse Act

Title IV of the Anti-Drug Abuse Act of 1986 (PL 99–570) authorized funding to address many of the needs identified in the IHS Alcoholism and Substance Abuse Action Plan. The Act required the development of a memorandum of agreement between the Departments of Health and Human Services and Interior, along with tribal consultation, to provide for a coordinated effort to address alcohol and substance abuse in American Indian and Alaska Native communities. That agreement was signed in March 1987 and identified both long- and short-term requirements and assigned lead responsibility for various tasks to either the IHS or the BIA.

Public Law 99–570 provided for the development of two specific types of youth services: A regional treatment center for acute detoxification and rehabilitation in each IHS Area, with the Phoenix and Tucson Areas to share a center, and community-based rehabilitation and followup services within each IHS Service Unit.

Two regional youth treatment centers have opened—one in Tahlequah, OK, and the other at the Acoma-Canoncito-Laguna Service Unit, NM. The remaining nine are in various stages of development, depending upon the special plans and programs designed by the IHS and the tribes in each Area. Each center will have an average of 24 beds for adolescents. The expected length of stay is 45–90 days. Individualized, multidisciplinary assessment and treatment plans with a family component and aftercare coordination will be emphasized.

School- and Community-Based Prevention Programs

Curriculums

Here's Looking at You: Original materials that were produced by the Comprehensive Health Education Foundation, 20832 Pacific Highway South, Seattle, WA 98198-5997, have been revised as "Here's Looking at You 2000" by Roberts, Fitzmahan and Associates. This complete drug education curriculum begins in kindergarten and continues through high school. Address: Roberts, Fitzmahan and Associates, 9131 California Ave., SW, Seattle, WA 98136, telephone (206) 932-8409.

Project Charlie: Chemical Abuse Resolution Lies in Education (Charlie) is a Storefront/Youth Action drug prevention program originally funded through the Community Education Training Act. Address: Project Charlie, 5701 Normandale Road, Edina, MN 55424.

BABES: Beginning Alcohol and Addiction Basic Education Studies (BABES) was developed by the National Council on Alcoholism (NCA) in Detroit, MI, in 1978. It is available through any NCA affiliate. Address: NCA, 17330 Northland Park Court, Southfield, MI 48075.

Community Programs

TRAILS: Testing Realities and Investigating Life Styles (TRAILS) is a program of alternative activities for youth. Contact: Ms. Nancie Young, Division of Community Services, Department of Health and Social Services, 1 W. Wilson St., P.O. Box 7851, Madison, WI 53707.

Circle of Life: Circle of Life is an annual training seminar in Michigan for Indian and non-Indian service workers. It is sponsored by tribes and financed with Federal and State funds.

Children Are People: Children Are People is a program that deals with sociological-emotional implications of drug abuse. Address: Children are People, Inc., 1599 Selby Ave., St. Paul, MN 55104.

> *'. . . it is clear that alcoholism and its secondary effects play a preponderant role in the overall mortality of American Indians.'*

Hiring of community rehabilitation staff at tribal and IHS facilities continues. More than one-half the needed skilled personnel have now been placed on duty. In addition, community education and training of staff have resulted in more than 5,000 persons being trained in basic concepts of alcohol and substance abuse and the special needs for prevention activities directed at youth. These efforts are continuing.

A unique provision of the Act was the authorization and funding to establish community-based programs for health promotion and disease prevention. Ten programs were competitively awarded funding in fiscal year 1987, covering activities such as teen clinics, fitness programs, and programs targeted at the prevention of alcohol and substance abuse among young Indians.

Tribal Activities

In the past, a major barrier to improving alcoholism prevalence rates has been the practice of not interfering with individual decisions, sometimes manifested as apathy, on the part of Indian communities, and lack of community sanctions against alcohol abuse. It appears to have been common experience for family members, for example grandparents, to excuse unacceptable behavior and even facilitate it by paying bills, including bail, and placing responsibility on others rather than the alcohol abuser themselves. Native American noninterference has been described by Good Tracks (*5*).

Regardless of the source of the community's acceptance of alcoholism, a turning point may have been reached with the experience of the Alkali Lake community in Canada, along with the growth of health promotion concepts in Indian communities. It has been reported that the Alkali Lake Band of Indians addressed alcoholism as a major problem by mobilizing the community against it (*6*). This move was stimulated by a recovering woman and her husband who later became chairman of the band. An essential component appears to have been a declaration that the community would no longer tolerate a high rate of alcoholism. Although it is too early to ascertain whether such action is effective, an increasing number of Indian communities in the United States are concluding that only the community itself can eliminate alcohol abuse. An increasing number of organizations such as Students Against Drunk Driving have been formed, and public declarations have been made by tribes that they intend to become alcohol free. For example, the Cheyenne River Sioux Tribe recently passed this resolution (*7*):

> BE IT FINALLY RESOLVED, for the spiritual well-being of our children and families, and for the survival and strengthening of our Districts from this day forward, let it be known that the Council of the Cheyenne River Sioux Tribe have declared war on all that is associated with alcohol and drug abuse and strive for the goal that by the year 2,000 the Cheyenne River Reservation will be 100 percent drug and alcohol free.

Recently, a group of teenaged persons on the Cheyenne River Reservation demanded that the tribe take action to decrease the amount of alcoholism on the reservation. Such action, along with a positive response by the tribe, would have been unthinkable a few years ago. The general impression is that a distinct change in the attitude of many tribes toward alcoholism has occurred, and many are beginning to take actions directed at solving the problem. In some tribes, this has taken the form of requiring sobriety among persons elected to tribal office. The presentations made on local TV and radio stations mentioned earlier are another model of community prevention activities.

Discussion

Many of the recommendations contained in the IHS strategic plan have been acted upon. In yearly individual work plans for the senior managers of IHS, preventive activities, among them alcoholism prevention, are included. This element serves not only to emphasize the importance of the effort, but also to provide a method by which specific actions can be instituted and measured. Another result has been the announcement that the official policy of the IHS is that alcoholic beverages not be served at any official social function. This appears to have been an important gesture not only for employees but for Indian people as well, indicating that the IHS has taken the challenge of alcoholism seriously. The strategic plan has also placed the IHS in a favorable position to respond readily to the Secretary's Initiative and to the passage of the Anti-Drug Abuse Act.

The steady fall in the mortality rate associated with alcoholism indicates that the problem, once thought of as hopeless, is, in fact, lessening—and at a more rapid rate than for the United States' all races population. It is as important to focus on this fact as it is to focus on the statistic that the mortality rate among Indians is more than four times higher than that for the U.S. all races group. Death rates associated with alcoholism appear to be highest in the IHS Areas of Billings, Phoenix, Tucson, and Aberdeen. This inter-Area variation is seen for other diseases and conditions. Such variation provides opportunities for investigation and, of equal importance, it is a source of information useful in targeting activities to the communities with the greatest needs.

The present philosophy of the IHS concerning health interventions was expressed in the July–August 1987 issue of *Public Health Reports* (*8*). Attention is given to lowering specific morbidity and mortality rates for the population served by the IHS. This will be accomplished by ensuring that the IHS service population (*a*) has a general knowledge of a number of health factors, (*b*) reduces and, where possible, removes various risk factors, and (*c*) targets efforts to those conditions that are responsible for, or contribute the most to, years of life lost prematurely.

The efforts for controlling alcoholism fit very well into this philosophy. The survey of community- and school-based prevention activities (*3*) indicates that in addition to a gratifying number of existing activities, there is an excellent infrastructure involving the Head Start Program, BIA- and tribally-operated schools, and other community organizations and activities upon which to expand present programs.

Progress to date with the implementation of PL 99–570 suggests that intervention and treatment for Indian adolescents will facilitate the early arrest of alcoholism and other drug dependency and, consequently, prevent drug-related illnesses, injuries, and mortality. In addition, it will make possible the dealing with dependency before the adolescent becomes a parent, thereby breaking the intergenerational cycle present in so many Indian families. The treated adolescent may be returned to the educational system during the life-stage when it is easiest to develop knowledge and skills necessary for a healthy, productive adulthood. Finally, dealing with adolescents will afford an opportunity to intervene with parents and family members.

Rehabilitation and aftercare services are essential to the proper functioning of a program for adolescents. Such services permit the coordination of efforts by a multidisciplinary team to screen and appropriately place Indian youths in inpatient or outpatient programs for treatment of chemical dependency. Case management, including collateral services for the youth and family and identification of Indian children whose primary problem is behavioral or educational, rather than substance abuse, will also be possible through aftercare services.

Obviously, success will not be achieved except by continued efforts applied over time. The IHS is committed to this approach and has declared its intention to control alcoholism among American Indians and Alaska Natives by the year 2000. Success will depend upon the Indian communities being committed to eliminating alcoholism. As stated in recent testimony before the Senate Select Committee on Indian Affairs, ". . . when the [American Indian/Alaska Native] communities take responsibility for their own wellness, especially as it relates to alcoholism and substance abuse, IHS and other efforts in the provision of resources and expertise will have a far greater benefit" (*9*). The increasing number of tribes that appear to be prepared to undertake this important responsibility is reason for substantial optimism, indeed.

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Biological Features of Women's Alcohol Use: a Review

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Dr. Roman is Professor of Sociology and Research Center Director with the Institute for Behavioral Research, University of Georgia. This paper was adapted from a 104-page review entitled "Women and Alcohol Use: A Review of the Research Literature" which focused on studies published in substance abuse research journals from 1980 to 1988 concerning the effects of alcohol consumption on women. The review was prepared while Dr. Roman served as consultant to the Division of Basic Research of the National Institute on Alcohol Abuse and Alcoholism. Tearsheet requests should be sent to him at the university, Rm. 548 GSRC, Athens, GA 30602.

Synopsis

Sensitivity to gender issues in the research community has generated a modest but growing amount of data on the biological effects of alcohol consumption on women. Data generally indicate that the same amounts of alcohol have greater effects on women and that women develop more severe alcohol problems than men over shorter drinking histories. Despite a number of studies, however, there are no clear differences between women and men in the impact of alcohol consumption on cognitive processes.

Although the findings are mixed, the data point toward greater physiological deterioration among women as compared with men who have similar drinking histories. These differences may be related to the differences in patterns of social recognition and reaction that occur in instances of alcoholism among women. Such differences are confirmed by other data that indicate greater social isolation and general disorganization among female alcoholics than among male alcoholics.

The risks of fetal alcohol syndrome that are associated with heavy alcohol consumption among women during pregnancy have been established, and a complex of other relationships between alcohol consumption and reproductive-related systems and behaviors exists. Linkages between sexual dysfunction, sexual satisfaction, and alcohol consumption appear to exist, but have not yet become clearly understood.

It appears that alcohol may be used as a self-medication to cope with perceived problems of sexuality. It also appears that heavy alcohol consumption can contribute to sexual dysfunction and dissatisfaction. A growing body of sophisticated experimental research has established relationships between patterns of alcohol metabolism and phases of the menstrual cycle, with this literature offering some of the clearest indications of distinctive differences between the sexes in the biological consequences and correlates of alcohol consumption.

THAT RESEARCHERS HAVE BECOME conscious of women's alcohol-related problems is evidenced by the growing number of studies concerning the sex-specific ramifications of alcohol use. The review that follows is limited to published reports concerning the effects of alcohol consumption on women compared with men.

Physical and Neuropsychological Effects

In addition to reporting higher levels of problems associated with heavy drinking, women also report higher levels of intoxication than men when consuming the same amounts of alcohol. This effect can be explained in part by the differences between the sexes in total body water content. Shortly after alcohol is consumed, it becomes uniformly diffused in both extra- and intra-cellular body water. Since tissue alcohol concentration is directly proportional to tissue water content, persons with lower total body water quantities will have a greater tissue alcohol concentration (*1*).

Smaller and fatter people have smaller body water compartments than larger, or leaner, more muscular people. Because of their higher percentage of fatty tissue, women have smaller body water quantities than men of comparable size, resulting in higher blood-alcohol concentrations in women. Moreover, there is evidence that women develop higher blood alcohol levels for given amounts of alcohol when it is ingested at the time of ovulation or just before menstruating (*2*). These effects are important considerations in epidemiologic research and in recommendations about safe drinking. Researchers have established that once differences in body water content are considered, there is no evidence of differences in the metabolism of alcohol between the sexes (*3*), nor are there differences in the physiological response to the effects of alcohol ingestion (*4*).

Further, evidence exists that chronic alcohol abuse has a greater physiological impact upon women than upon men. Alcoholic women are more frequently disabled and are disabled for longer periods than male alcoholics—reflecting, in part, the differences in identification processes that, in turn, embody sex-related social norms. The percentages among women alcoholics who die from such causes as suicides, alcohol-related accidents, circulatory disorders, and liver cirrhosis are much higher than among men alcoholics. Several international studies show consistently that female alcoholics have death rates from 50 to 100 percent higher than those of male alcoholics (*5*).

A longitudinal study of 103 American women alcoholics followed for 7 years after their treatment in 1967–68 revealed that nearly a third had died, a rate 4 1/2 times higher than expected. The average life span of these women was decreased by 15 years. Women who were older when they sought treatment, who became alcoholic before age 30, or who had histories of frequent binge drinking were most likely to have died prematurely; those who became abstinent were the most likely to have survived (*6*). Thus, a common finding across many studies is that women have shorter drinking histories but similar or worse consequences than men even though the absolute amount of alcohol consumed during the history may be similar.

Many ramifications of using alcohol are associated with the effects it has on the cognitive and motor functions of both men and women. Given that differences have been found between the sexes concerning other physiological consequences of using alcohol, the amount of attention given to researching the differences in the effects of alcohol on neuropsychological functioning is modest. One study comparing neuropsychological functioning between alcoholic women and men in treatment indicated women to be significantly more impaired (*7*).

This difference was confirmed in research by Acker (*8*), who compared the neurological deficits between female alcoholics and non-alcoholic controls. The expected differences were found, and a comparison of this research with previous studies of male subjects showed that the performance deficits among males were comparable to those among females, even though the drinking histories of the female alcoholics were considerably shorter.

Using a comprehensive battery of cognitive tests, Crawford and Ryder (*9*) reported a British study of 8 male and 8 female alcoholics who had completed treatment in an inpatient unit that revealed similar patterns of cognitive impairment between the men and women. Similarity in cognitive impairment was found despite the significantly shorter history of heavy drinking and lower levels of routine consumption among the females—again indicating greater impact per dosage unit among females as compared with males.

Meier and coworkers (*10*) studied the accuracy of recall of alcohol consumption among men and women college students and found a significantly lower accuracy of recall among the men. The authors explained this unexpected findings on possible differences between men and women in the effect of alcohol on short-term memory or the greater experience in drinking among men, compared with women.

Hesselbrock and coworkers (*7*) reported a significant interaction between the sexes and cognitive performance among alcoholics with antisocial personalities, with women performing cognitive tasks at lower levels than women without antisocial personality diagnoses. A study reported by Carey and Maisto (*11*) of the effects of drinking on cognitive processes of female social drinkers did not, however, confirm an effect of alcohol consumption on cognitive processes. Sixty female drinkers were randomly assigned to abstinence or drinking groups, with the results of a battery of cognitive tests indicating no distinctive differences between the groups. This led the authors to conclude that recent drinking practices were not related to cognitive test performance among this sample of female social drinkers.

In a thorough and detailed review of the relationship between drinking and cognitive functioning, Parsons (*12*) urges caution in drawing conclusions about what many researchers have come to assume is a direct causal effect between drinking and impaired cognitive performance. His review of the research reveals frequent differences between the sexes when both men and women are included in experiments. Also, there are some hints that the emotional state accompanying drinking may affect cognitive outcomes. Such a relationship may explain, in part, the differential effects between the sexes, given a large amount of other data confirming differences between the sexes in emotional and attitudinal orientations to drinking. At the same time, there is no conclusive biological research to account for possible differences in the cognitive or neurological impact of drinking between the sexes.

In addition, there is growing evidence of differences between the sexes in the physiological impact other than in brain functioning. Evidence from treatment-based research indicates that women may experience greater physiological impairment than men early in their drinking careers, despite having consumed less alcohol than comparable men; advanced liver disease was more frequent (*13*), and alcohol-related illnesses in general were more frequent among women despite drinking histories similar to men in the samples (*14–16*).

Mortality

Mortality data (*6*) for a group of 100 hospitalized women alcoholics (57 percent primary, 8 percent secondary, 25 percent affective disorder, and 7 percent sociopathic alcoholics), who were followed for 12 years after hospitalization, showed that 30 percent of them had died by the end of followup. Based on life expectancy at hospitalization, the women lost an average of 25.9 years because of alcoholism (mean age at depth = 51). One-third of the deaths were due to cirrhosis and one-fifth to unnatural causes (suicide, accidents, and homicide). Forty-one percent of the natural deaths were sudden and occurred unexpectedly. The highest mortality rate occurred in the group diagnosed as sociopathic alcoholics, with over one-half dead at followup (mean age 39.8). Only 1 of the 8 secondary alcoholics died. Mechanisms underlying these differences are unknown, but may be related to estrogen (*17*) or sex-related immune response (*18*).

A long-term followup of 1,312 alcoholic patients referred to a Swedish hospital during 1949–69 was reported to Burglund (*19*). The study included an assessment of causes of death and the relationship of symptoms revealed during treatment to the cause of death. In that study, which comprised 120 women, there were no significant differences between the men and women alcoholics in terms of deaths revealed during the followup research period.

Other Comparisons

Possible physiological differences between women and men are indicated in a study reported by Connelly and coworkers (*20*). The subjects were 12 female and 12 male nonalcoholic heavy drinkers who were otherwise physically healthy. The data revealed significantly greater glucose intolerance among females, compared with the male subjects. The results suggest that there may be significant differences between women and men in the metabolism of alcohol. A thorough review of the research literature by Dancy and Maxwell (*21*) indicates that there is no strong evidence for differences between the sexes in terms of the effects of alcohol on cardiomyopathy.

In a detailed review of the published literature, Johnson and Williams (*22*) report that it is clear that females develop more severe liver disease at a lower mean daily alcohol intake and after a shorter duration of drinking than do males. They explain this difference on the differences in body composition associated with body size, fluid content, and hormonal distinctions.

Further insight into the differences between the physical consequences of alcohol consumption for men and women is revealed in a study by Mendelson and coworkers (*23*), which focused upon medical disorders among 10,758 consecutive admissions for inpatient alcoholism treatment. The rates of medical problems in addition to alcoholism among men and women were very similar. In terms of specific diseases accompanying their alcoholism, women were somewhat more likely to exhibit neuroses and other psychiatric disorders; men were substantially more likely to display hypertensive disease and other forms of circulatory disorder. Diseases of the liver, gall bladder, and pancreas, as well as cirrhosis, had very similar rates for men and women.

The possibility of differential effects of heavy drinking among men and women receives some support in a study reported by Barrison and coworkers (*24*). In contrast to men, a significant correlation was found between mean corpuscular volume, liver size, and alcohol consumption. The subjects in the study were self-referred heavy drinkers who were members of Drink Watchers and thus were believed to be persons highly concerned about their drinking. Because other physiological characteristics and drinking histories were similar, the researchers were led to conclude that, among heavy drinkers, females were likely to develop liver problems more rapidly than males.

In a study of 99 men and 57 women who had been diagnosed with alcohol-related liver disease, Saunders and coworkers (*25*) found that women were significantly less likely than men to have been advised by others to reduce their alcohol consumption, to have received previous hospital treatment for alcohol problems, to have received counseling for alcohol problems, or to have been convicted for any alcohol-related offenses. The women were also less likely to have been advised to stop or reduce their drinking by family members, or to have been advised to stop or reduce drinking by a general practitioner, but were equally likely to have attended Alcoholics Anonymous and substantially more likely to have received psychiatric treatment for nonalcohol-related problems. The researchers observed that the alcohol dependence syndrome of most of these patients was relatively mild, but the differences between the sexes in the pattern of identifying the patients' alcohol problems indicate different norms utilized by both the lay public and professional practitioners. The specific focus of the study also indicates that the lower efficacy of social reactions toward alcohol-dependent women may indirectly increase their risk of liver disease.

In a study of the flushing response to alcohol use among Koreans and Taiwanese, Park and coworkers (*26*) found significantly more flushing among Korean women compared with Korean men, but no differences were found between the Taiwanese men and women. The researchers cannot explain these results, but they suggest that differences in body weight may be the key.

Effects on Reproductive System

Alcoholism is associated with high rates of sexual, gynecologic, and reproductive problems among women, paralleling problems of impotence and sterility among male alcoholics. Seventy percent of alcoholic women in one study reported sexual dysfunction (*27*). In another study of 30 alcoholic women, 75 percent reported they experienced decreased sexual satisfaction after drinking became a problem, together with difficulty in many instances of achieving orgasm. Nearly half had become anorgasmic (*28*).

In a recent study of 55 alcoholic women who were matched with 54 nonalcoholic women, it was found that for women alcoholics sexuality and partner relationships are definitely more stressful and conflicting—symptoms categorized by the researchers as "disorders of sexual desire"—than for the nonalcoholics (*29*). The authors suggest that the model of Helen S. Caplan provides a useful explanation. This theory suggests that maternal-related problems, anxiety, and conflicting norms produce phobic avoidance of sexual encounters or the creation by women of destructive erotic atmospheres that, in turn, produce disorders of sexual desire—with all of these processes interacting with the effects of alcohol.

In a national survey of 917 women, Klassen and Wilsnack (*30*) report that heavy drinking is correlated with dysfunctional sexuality and hypothesized that this may be a vicious circle, which some of their respondents apparently escaped by abstention. They also found that suppressed and traditional sexuality is more frequent among those who are lighter drinkers and abstainers. Most women drinkers reported that drinking lessens their sexual inhibitions and helps them feel close to others; this consequence occurred most frequently among women who drink heavily. More than half the sample reported that they have been targets of other drinkers' sexual aggression while drinking. A sexual dysfunction index reveals that moderate drinkers scored significantly lower than both lighter and heavier drinkers. The heaviest drinking women had the highest rates of sexual disinterest over their life time and also were most likely to report the lack of orgasm. The researchers also reported that nontraditional sexual behaviors were more likely to occur among heavier drinkers. The potential efficacy of drinking in its relationship to sexual behavior is indicated by elevated sexual dysfunction rates among women who were abstainers but who had substantial drinking histories.

Harvey and Beckman (*31*) studied the relationship between alcohol consumption and female sexuality by having 69 women maintain logs of drinking and sexual behavior. The data indicate that female-initiated sexual activity was greater with less alcohol use, although the questionnaire data indicated that the study subjects believed alcohol enhanced their sexual desire, enjoyment, and activity. In terms of risk-taking behavior, the data indicated that alcohol consumption prior to sexual intercourse did not significantly alter the use of contraceptives.

An examination of the relationship between drinking and reported sexual behavior in a comparative study of alcoholic and nonalcoholic women revealed that the alcoholic women were significantly more likely to report enjoyment of sex while drinking, were more likely to engage in intercourse after drinking, but overall reported a lower level of sexual satisfaction (*32*). A review of literature related to sexuality notes a paradox between the physiological depressive effects of alcohol on sexuality coupled with women alcoholics' apparent increase in subjective arousal. Reviewing the exten-

'The findings indicate that repeated or sustained episodes of alcohol intoxication may suppress hormonal activity in women.'

sive materials on alcoholic women's sexual dysfunctions, the author concludes that women may use alcohol to self-medicate the sexual difficulties they experience (*33*). A comparative study of 34 alcoholic and 34 nonalcoholic women revealed that the alcoholics reported more guilt related to sexual activity and significantly less control over their sexual lives (*34*).

Thus the empirical evidence indicates an association between alcohol consumption and sexual function, but the direction of the relationship is unclear. The available evidence definitely points toward a circular pattern of causality, with alcohol affecting sexual function and reactions to sexual dysfunction possibly encouraging increased use of alcohol, or situation-specific use of alcohol vis-à-vis sexual encounters. In addition to these interactions, there are likely interactions between women's sexual activities in relation to alcohol and those of male partners who also may be consuming alcohol; this subject remains to be investigated extensively.

In terms of gynecologic dysfunction, a 1981 national survey revealed a positive correlation between women's drinking levels and their reports of dysmenorrhea, heavy menstrual flow, and premenstrual discomfort (*35*). There was a particularly strong association between these disorders and the report of at least one 6-drink-or-more episode per week. Women who averaged three or more drinks per day or who experienced at least five events of six drinks or more per week had higher rates of most types of gynecologic surgery. Finally, a history of miscarriage or stillbirth, premature birth, birth defects, and infertility was found to be associated with highest levels of alcohol consumption.

It may not be correct to conclude that there is an overarching relationship between gynecologic- and reproductive-related functions and alcohol consumption. Working to measure the effects of moderate amounts of alcohol on cognitive and physiological functions during different phases of the menstrual cycle, Brick and coworkers (*36*) found no significant differences in behavioral effects across different menstrual phases.

However, Youcha (*37*) summarizes the data dealing with the relationship between drinking and the menstrual cycle: after a drink, women's reactions are slower just before her flow begins, at the midpoint, and during the flow than they are during the rest of the month.

Influence of the menstrual cycle on drinking behavior is confirmed by the study of 69 normally menstruating women reported by Harvey and Beckman (*38*). Through subjects' personal monitoring of drinking behavior, the researchers found that frequency of alcohol use did not vary among the five phases of the menstrual cycle but that quantity of alcohol consumption significantly fluctuated and peaked during the luteal phase of the cycle. Although there is substantial evidence from other research that there may be interaction between hormonal fluctuations and reactions to alcohol consumption, these investigators suggest that such metabolic differences may influence the choice behavior of women regarding drinking at different phases of the menstrual cycle.

In a study of acute response to equal dosages of alcohol among 11 male and 13 female social drinkers (*39*), females were found to consistently achieve higher blood alcohol levels. This result was explained mainly by the preponderance of the women being in the middle stage of the menstrual cycle. Women not using birth control medication also attained higher blood alcohol levels than the males. Very few cognitive or psychomotor distinctions were found between the two groups, with the exception of the men recovering memory function more quickly on the descending limb of the blood alcohol curve. The stage of the menstrual cycle was not found to affect psychomotor or cognitive performance while the women were intoxicated.

In an experimental study of 16 men and 18 women, Wilson and Nagoshi (*40*) reported differences between the men and women in the consistency of data on alcohol metabolism, sensitivity to alcohol, and acute tolerance to alcohol, indicating significantly more day-to-day variability in reactions to alcohol among women, as compared with men. That study did not indicate significant effects of menstrual cycle phases on the alcohol metabolism data.

In a carefully designed double blind study, Mendelson and coworkers (*41*) found significant effects of alcohol on female hormone secretion, controlling for the phase of the women's menstrual cycle. The findings indicate that repeated or sustained episodes of alcohol intoxication may suppress hormonal activity in women.

Substantiation of the difference in ethanol metabolism was found in an experimental study of 63 women and 75 men reported by Cole-Harding and Wilson (*42*). The data showed that women metabolized alcohol faster than men regardless of the phase in their menstrual cycle and whether or not they were taking oral contraceptives. The researchers concluded that these data indicate a lack of effects of female hormones on ethanol metabolism, leaving the explanation of male and female differences to other factors.

In a detailed study of the relationship between alcohol intoxication and the menstrual cycle, Sutker and coworkers (*43*) reported that differences in alcohol metabolism were found across different phases of the menstrual cycle among women who had ovulated during two previous cycles. Shorter elimination times, reduced areas under the blood alcohol time curve, and faster disappearance rate characterized the midluteal phase of the menstrual cycle compared with the early follicular and ovulatory phases—indicating that elimination was facilitated during the midluteal phase. Since this was the first study to measure serum level of estradiol progesterone and other hormonal levels over two consecutive menstrual cycles on each day of acute alcohol intoxication, the distinctive effects of the menstrual cycle on alcohol metabolism appear strongly confirmed.

These effects may not be sustained across the life cycle, however. Gavaler (*44*) underlines the importance of studying the effects of alcohol on endocrine function in postmenopausal women. She points out the associations between hormonal functions and alcohol consumption and the associations between hormonal functions and disorders that are most prevalent at postmenopausal ages, including cancers and osteoporosis. In her review of an extensive number of studies, Gavaler concludes that the effects of alcohol during the postmenopausal phase of life have not been adequately specified or understood and that considerable more research is needed.

More recently, however, Valimaki and coworkers (*45*) reported an experimental study of the effects of hormonal secretion of alcohol ingestion among postmenopausal women. Ten women were included in the study. The results indicated that there were no acute effects of alcohol ingestion on the secretion of the major gonadotropins in women. The authors interpret these findings as consistent with previous research.

Thus, although gynecologic, obstetric, and sexual problems appear to be linked to both alcoholism and alcohol abuse, the mechanisms of these associations are unclear. Both biological and psychosocial causes are probably involved. Impaired sexual functioning may be both a cause and a consequence of excessive drinking, underlying depression, and poor interpersonal relationships. Ironically, excessive drinking does offer a means of coping with inadequate sexual response and the anxiety surrounding it. This vicious circle may come into play in response to gynecologic and obstetric difficulties, as well as affecting sexual activity. The evidence of significant interactions between hormonal levels and alcohol consumption is compelling, and the sophistication of research that is focused on these relationships is increasing. At present, it appears that research does not offer clear guidance for women's drinking behavior relative to the menstrual cycle or to possible adverse impacts that may be associated with drinking in the postmenopausal period of life.

Research on the relationship between alcohol consumption and reproduction has had a rather intense but singular focus. The fetal alcohol syndrome (FAS) has received a great deal of attention during the past decade. Care should be taken when identifying this as a women's alcoholism issue. Media coverage, in particular, appears to place FAS responsibility on women, ignoring the barriers and difficulties that women may have in finding or receiving assistance for an alcohol problem. Further, the issue is often structured as if pregnancy were solely the responsibility of the pregnant female. Since other research clearly shows that people of both sexes are influenced by the drinking behavior of their spouses and interactional partners, it is clear that males have major responsibilities in controlling the effects of alcohol during a spouse's or partner's pregnancy. Furthermore, many cases of FAS involve symptoms that extend beyond the direct effects of the mother's drinking, especially the resources and care directed toward the infant in the postnatal period. Thus, care should be taken to avoid ''blaming the victim'' in considerations of FAS.

Adverse effects of alcohol ingestion by pregnant women and their offspring were first clinically suspected over 200 years ago, but only recently have those effects been more precisely specified. FAS neonates have three characteristics that have been associated with maternal alcohol use: growth retardation before or after birth; abnormal features of the face and head, such as unusually small head

Care should be taken when identifying fetal alcohol syndrome as a women's alcohol issue. . . . the issue is often structured as if pregnancy were solely the responsibility of the pregnant female.

circumference and flattened facial features or both; evidence of central nervous system abnormality, such as mental retardation. Whether FAS is the leading cause of mental retardation in the Western world (*46*) has been argued recently. Aside from the personal impacts, the social and economic costs are dramatic over the lifespan of the FAS victims.

Beyond those mentioned, other effects that have been identified as associated with maternal drinking during pregnancy are termed "fetal alcohol effects" (FAE). FAE include a range of congenital abnormalities such as eye and ear defects, heart murmurs associated with defective development of the heart chamber wall, genitourinary defects, hemangiomas, and fingerprint and palmar crease abnormalities.

Evidence based on animal and human research leaves little doubt that serious birth anomalies can result from alcohol ingestion, but the frequency with which they occur and the level of alcohol use that can cause them in humans is by no means certain.

Estimates of the worldwide prevalence of FAS have been centered at 1.9 per 1,000 live births (*46*). In the United States, epidemiologic research has revealed especially high prevalence among American Indian groups, ranging in one study up to 9.8 per 1,000 live births for certain Plains Culture tribes, this being the highest rate found anywhere in the world (*47*). Alcohol-related effects of lesser severity in the infants of heavily drinking women may occur as often as in 69 percent of live births. Little reported a 91-gram decrease in birth weight associated with about two drinks (1 oz. of pure alcohol) per day consumed by the mother before she knew she was pregnant. A 160-gram deficit was found in infants of women who drank that much during the seventh to ninth months of pregnancy (*48*). A more recent study found that drinking an average of two or more drinks a day tripled the likelihood of preterm delivery (*49*).

Unfortunately, there are many problems with such dose-effect estimates. Alcohol dependent women may be inclined to understate the amount of their drinking. This may be especially true if they feel guilty about drinking while pregnant. Other factors such as lifestyle, smoking, other drug use, poor diet, or poor general health may both cause and interact with alcohol use during pregnancy in producing problems that may affect neonatal health. Paternal drinking is known to reduce sperm counts and sperm motility and to produce other sperm abnormalities. That the father's drinking may also contribute to fetal abnormalities cannot be dismissed, although the limited studies that have been done have not found such an effect (*50, 51*). Experimental administration of alcohol doses to pregnant women while carefully controlling for other possible factors is not, of course, ethically possible.

The significance of maternal abstinence during pregnancy is underlined by research reported by Coles and coworkers (*52*). These investigators examined the behavior of 103 neonates born to three groups of women: those who drank a mean of 12 oz. of alcohol per week throughout pregnancy, those who drank a mean of 14 oz. per week but stopped during the second trimester, and those who never drank during pregnancy. The research population was composed primarily of black women of low socioeconomic status.

The data revealed that infants exposed to alcohol any time during gestation had significant alterations in reflexive behavior, less mature motor behavior, and an increased activity level. Compared with infants whose mothers did not stop drinking during pregnancy, neonates whose mothers had stopped during the second trimester were superior to those whose mothers continued to drink in terms of need for stimulation, motor tone, tremulousness, and problems in reflexive behavior. The authors conclude that exposure to alcohol during any phase of pregnancy affects the neonate, although cessation clearly reduces those effects.

In a study of drinking practices among pregnant women, Stephens (*53*) found that more frequent high maximum drinking and increased drinking during pregnancy were primarily found among blacks and among teenage mothers, indicating that the risk for adverse fetal outcomes may be particularly high in the subgroup with both of these characteristics.

In another report from that study of 311 metropolitan prenatal patients (primarily black) in a southern city, Stephens (*54*) reported that "social support" was significantly associated with decreased alcohol consumption during pregnancy.

Disaggregating social support, the researcher reported that specific pregnancy support worked directly against drinking that might have damaged the fetus, whereas other social support, involving friendship networks, might operate to enhance drinking activities. Thus, caution should be exercised in recommending social support as a means of facilitating appropriate drinking behaviors among pregnant women. A similar pattern indicating the particularly strong influence of friends, spouses, and sexual partners on the drinking behavior of black women was reported in a study by Fernandez-Pol and coworkers (*55*).

Smith and coworkers (*56*) collected data from 267 women before and after they gave birth to ascertain the predictors of those who continue to drink and those who stopped drinking during pregnancy. The data revealed that the offspring of the women who continued to drink were found to exhibit a higher frequency of growth retardation and neurobehavioral and structural consequences compared with the infants of nondrinkers or of mothers who discontinued using alcohol during pregnancy. Many of the infants showed evidence of withdrawal syndrome. The data comparing the two groups revealed strong distinctions on the extent to which the women who continued to drink were characterized by definable drinking problems. The findings suggest that women who continue to drink during pregnancy may be appropriate targets for intervention focused on alcohol abuse, and that this continuing behavior may reflect their being embedded in alcohol problem syndromes rather than being unresponsive to information indicating the risk associated with drinking during pregnancy.

It is critical to point out, however, that the number of FAS cases falls far short of the estimated numbers of pregnant women who drink abusively (*57*). This statistic indicates the possibilities of both physiological protectors and vulnerabilities that are the continuing focus of investigation. ''Social'' drinking is not without its impacts, however. A carefully designed study in which 84 drinking pregnant women were included only if their consumption remained within typical ''social'' norms revealed distinctive decrements in their children in terms of language and verbal comprehension (*58*). On the positive side, a recently reported study indicates that women are increasingly conscious of the possible adverse effects of drinking during pregnancy and reduce or terminate their consumption accordingly. Although many recommendations center on abstinence during pregnancy, that study found that fewer than two drinks weekly had no detectable adverse effects on the fetal outcome (*59*).

As with other teratogens, the question of just how much is needed to affect the fetus at different points in development is also unclear. Taken at a point of maximum fetal toxicity, alcohol may kill the fetus, resulting in a spontaneous abortion that may go undetected (or unreported) in the individual case. At other points, either because the individual fetus is genetically more resistant to damage or the stage of development makes damage less likely, there may be no detectable alcohol-related anomalies.

Animal studies provide some indications of possible human effects. One such study designed to resemble human ''binge drinking'' used the animal equivalent of two large doses of alcohol given on a single day during gestation. The resulting damage to the mouse fetuses closely resembled that found in cases of human FAS (*60*). Other animal studies of alcohol's effects have duplicated such behavioral abnormalities in human offspring as hyperactivity and learning problems presumably caused by alcohol use during pregnancy (61–63).

Summary

There is some evidence that biological differences between the sexes may lead to the greater effect that alcohol has on women than men, both in terms of more rapid intoxication and in terms of deterioration that accompanies histories of alcoholic drinking. The menstrual cycle appears to interact with alcohol metabolism and, thus, with the behavioral effects of drinking. There is an impact of alcohol on women's gynecologic functioning, with much more to be learned about the mechanisms involved and their implications. FAS is a major problem associated with women's drinking behavior. Women's drinking during pregnancy can affect their offspring, although the prevalence of the FAS is notably lower than the rates of heavy and abusive drinking among pregnant women. Further research may substantiate current findings that even modest alcohol consumption during pregnancy may be correlated with adverse outcomes during later phases of child development.

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"Fetal Alcohol Syndrome (FAS) is among the three leading causes of birth defects with accompanying mental retardation, and the only preventable one among the top three. To avoid development of alcohol-related birth defects, the best advice to pregnant women is not to drink."

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Alcohol-Related Birth Defects: an Update

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Synopsis

Historically, mankind has at least suspected that alcohol was somehow connected with undesirable effects on progeny. In the 18th century, physicians became aware that maternal alcohol consumption resulted in excess fetal and neonatal mortality, low birth weight, and many other deleterious effects. Perhaps as a response to the temperance and Prohibition periods, scientists lost sight of or interest in the effects of alcohol in pregnancy. In the late 1960s and early 1970s, the issue surfaced again, and scientists began systematic and in-depth studies of fetal alcohol syndrome (FAS) and alcohol-related birth defects (ARBD).

Epidemiologic research now suggests that FAS has outranked Down's syndrome and spina bifida in prevalence and is now the leading known cause of mental retardation. Further, it is the only one of these three that is preventable. Because a safe limit of alcohol consumption in pregnancy is not defined, abstinence during pregnancy is the most prudent preventive measure.

Factors such as race, beer drinking, maternal weight gain, and low socioeconomic status are associated with a statistical increase in the incidence of FAS. In families where one child has been diagnosed as having FAS, the incidence rate can be as much as 405-fold higher than the worldwide average.

Neurobehavioral deficits can occur in the offspring of drinking mothers in the absence of a diagnosis of full FAS. The deficits differ with age and seem to persist into adulthood. Mental retardation or borderline mental retardation is a nearly ubiquitous neurological deficit in diagnosed FAS. In one study, it occurred in 75 percent of the non-FAS offspring of mothers who continued to drink heavily throughout their pregnancy.

From the mid-1970s, having established that alcohol is a teratogen, scientists have been trying to uncover the mechanism by which alcohol exerts its embryotoxic effects. Recent promising neuroanatomical studies have demonstrated that alcohol has a deleterious effect on neuronal migration and hence on the development of the cerebral cortex. Other studies have shown that prenatal alcohol exposure, by adversely affecting hippocampal development, may be responsible for the learning deficits so frequently encountered in FAS children. Other research has implicated prostaglandins in the mechanism of alcohol-related dysmorphology.

HISTORICALLY, there have been various cautions and proscriptions concerning alcohol and procreation. In the 18th century, London presented a unique opportunity to observe alcohol's effects on progeny. In a comprehensive review of historic literature, Coffey describes what is commonly known as the "Gin Epidemic," from 1720 to 1750 (*1*). In an effort to support the grain-producing gentry, Parliament reduced controls on distilling and selling of gin, expressly to promote its production and consumption. Public houses abounded, and the sale of gin flourished. Observations of excess fetal and infant mortality at ages less than 5 years were attributed to the consumption of alcohol (*2*). Birth rates declined, and death rates increased in a period of good wages, plentiful food, and relative freedom from epidemic diseases. More than a few reports and petitions by physicians, health care providers, and legislative committees speak of effects on children that correlate with some of the features of fetal alcohol syndrome (*2*). For example, in 1725 the College of Physicians in London warned the House of Commons that alcohol is "... too often the cause of weak and feeble, distempered children" (*3*).

Observations and reports of alcohol's effects on the growth and development of children continued throughout the 18th and 19th centuries; often they were a mixture of medical science and temperance mortality (*1,2*). In one 1899 paper, a carefully elaborated study for that period, Dr. William Sullivan recorded his observations of 600 children born of 120 female alcoholic prisoners in a Liverpool prison (*4*). Sullivan's work represents one of the earliest descriptions of what was later to be called fetal alcohol syndrome.

With the advent of Prohibition in 1920, the issue of alcohol's effects on pregnancy outcome was largely ignored both in the United States and Britain for nearly 20 years—until 1940, when new research again began to emerge. Much of that research consisted of animal studies that ridiculed the moralistic studies of the pre-Prohibition era (*2*) and attempted to refute the "germ cell damage" caused by alcohol (*5*). Indeed, a great deal of the medical writing up to and into the sixties not only condoned maternal alcohol use, but even prescribed it for "sluggish digestion" (*6*) and frequently for delaying labor.

In 1967, a family practitioner from Rennes, France, Dr. Alexandre Lamache, spoke before the French Academy of Medicine concerning his observations—throughout his 37 years of practice—of 1,245 children of alcoholic parents (*7*). The importance of that report does not lie in its adherence to any scientific rigor, but in its signaling a renaissance of interest in alcohol-related birth anomalies. His observations included neurological dysfunction, mental retardation, behavioral disorders, genital malformations, facial anomalies, and excess infant mortality. In 1968, also in France, Lemoine and his coworkers published what seems to have been the first well-organized study of alcohol-related birth defects among humans in the modern era (*8*). In the United States, the era of interest in fetal alcohol effects was presaged by a report by Ulleland in 1970 on prenatal growth deficiencies in 10 children of alcoholic mothers (*9*). Eight of these children were examined by Jones and Smith, who recognized a distinct pattern of abnormalities, a syndrome, in four of them. In 1973, they and their colleagues reported their finding, which they called fetal alcohol syndrome (FAS) (*10*).

Today, alcohol is recognized as a teratogen, that is, a substance that is capable of producing birth defects (*11*). The consequences of alcohol use in pregnancy range from subtle neurological disturbances and reduced birth weight to the unique cluster of abnormalities that constitutes FAS. To meet the diagnostic criteria set by the Fetal Alcohol Study Group of the Research Society on Alcoholism (*12*), a diagnosis of FAS requires specific manifestations in three areas: (*a*) prenatal or postnatal growth retardation (below the 10th percentile), (*b*) central nervous system perturbations that may include tremulousness, poor sucking reflexes, abnormal muscle tone, hyperactivity, attentional deficits, or mental impairment, and (*c*) at least two characteristic facial anomalies including narrow eye width, ptosis, a thin upper lip, a short upturned nose with underdevelopment of the groove between the base of the nose to the top of the upper lip, and general underdevelopment (hypoplasia) of the midfacial area. Two other terms "alcohol-related birth defects (ARBD)" and "possible fetal alcohol effects (FAE)," have been used in reports on alcohol and pregnancy outcome to address the consequences of prenatal exposure to alcohol that do not meet the minimal FAS criteria. ARBD are complications of pregnancy or birth defects that are attributable to alcohol after statistical analysis has corrected for the contribution of other possible factors (*13*). Possible FAE are birth defects observed in children of women known to have consumed significant amounts of alcohol in pregnancy that are likely, though not definitively, attributable to alcohol use (*14*).

Prevalence

Exact prevalance figures for fetal alcohol syndrome are not available, but the best estimate—based on local reports from various U.S. and European studies—is a range from 1 to 3 cases per 1,000 live births (*11*). Abel and Sokol, basing their estimate on 20 studies from Australia, Europe, and North America, found a worldwide incidence of 1.9 cases of FAS per 1,000 live births (*15*). At this rate, FAS is the leading known cause of mental retardation, ranking ahead of Down's syndrome and spina bifida (*16*).

Certain factors seem to influence incidence rates for FAS. When one studies the population most at risk for FAS—children of pregnant women who abuse alcohol or are alcoholic—the incidence rate rises dramatically. Abel and Sokol found that the incidence rate of FAS among mothers who were alcohol abusers was 59 per 1,000 live births, based on 14 U.S. studies that identified the incidence of alcohol abuse during pregnancy (*15*).

Abel reviewed literature on more than 300 clinical case studies in which siblings were mentioned to determine the incidence of FAS and ARBD among younger and older siblings of an FAS patient (*17*). Among older siblings, he found an incidence rate of 170 per 1,000 live births for FAS and 417 per

1,000 for ARBD. Among younger siblings of the index patient, the incidence of FAS was 771 per 1,000 and 886 per 1,000 for ARBD.

From two studies of more than 13,000 pregnancies, it was found that, in addition to persistent exposure to alcohol, susceptibility to FAS and ARBD increases with these factors: black race, frequent beer drinking, lower maternal weight and weight gain, and low socioeconomic status (*15,18*). In a study on the epidemiology of FAS among Indians in the Southwest, May and his colleagues found that being part of a Southwest Plains Indian culture also may lead to increased susceptibility to FAS, with an incidence of 9.8 per 1,000 births (*19*). An even higher occurrence of FAS was reported by Robinson and his coworkers (*20*) for a small isolated Indian community in British Columbia. In contrast, May and coworkers found that FAS incidence rates among Pueblo and Navajo Indians were more in line with the worldwide figure of 1.9 cases per 1,000 live births (*19*).

In a study to determine the critical period in gestation when alcohol causes anomalies, data on 359 neonates who were prenatally exposed to alcohol were subjected to multivariate analysis. The analysis confirmed that the critical period for alcohol teratogenicity is around the time of conception (*21*).

Neurophysiological Effects

Examples of some of the neurological problems that exist and persist in some children of drinking mothers have been described by Streissguth and LaDue (*22,23*). Among them are learning disabilities, speech and language problems, hyperactivity, and attentional deficits. Children with a diagnosis of FAS have an average IQ of 68–70.

Neurological problems resulting from prenatal exposure to alcohol apparently array along a continuum throughout the life of the affected person. By analyzing data from a large number of epidemiologic studies that reported neurobehavioral outcomes associated with prenatal exposure to alcohol, Streissguth found that the following neurological deficits accrue to the neonatal period: poor sucking, disrupted sleep states, low levels of arousal, tremulousness, unusual body orientation, excessive mouthing, abnormal reflexes, hypertonia, and poor habituation to redundant stimuli (*24*). Progressing to older infancy, one finds disrupted sleep-wake patterns, poor visual recognition memory, and decrements in mental and motor development, spoken language, and verbal comprehension. Preschoolers exhibit attentional deficits, delayed reaction time, and decrements in fine and gross motor performance. Adolescents present psychosocial problems and more overt psychopathology, and adults have problems in adaptive living and self-sufficiency.

Central nervous system (CNS) deficits were the subject of two Swedish studies of children born to alcoholic mothers. Aronson and Olegard studied 8- and 9-year-old children of alcoholic mothers and found that 25 percent attended special schools for the mentally retarded, and 35 percent had borderline mental retardation that required special help in school (*25*). Forty-three percent of the children of mothers who stopped drinking by midpregnancy and 75 percent of the children of mothers who continued to drink were mentally retarded or borderline mentally retarded. In a study by Larsson and Bohlin, 80 percent of children from alcohol abusing mothers presented behavioral disturbances or retarded psychomotor development or both (*26*).

Coles and coworkers conducted a series of studies on 103 neonates without FAS who were born to black mothers of low socioeconomic status (*27*). The infants were divided into groups whose mothers did not drink, who drank an average of 12 ounces of absolute alcohol (24 drinks) per week throughout their pregnancies, and who drank that amount but quit by the second trimester. Both alcohol groups were less optimal in neurobehavioral responses when examined at 3 days, but those whose mothers continued to drink throughout pregnancy were significantly lower in their orientation toward auditory and visual stimuli, motor performance, and autonomic regulation than those whose mothers did not drink. A subsample followed up at 30 days showed persistent behavioral deficits in the continued drinking group in reflexive behavior and autonomic control. An examination at 6 months of 60 of the infants found that differences in orientation, motor performance, reflexive behavior, and autonomic control at 3 days were predictive of mental development and motor performance at 6 months.

Streissguth likewise found persistent behavioral deficits in her cohort of children of heavier-drinking mothers to at least age 4 (*28*). The 4-year-old children whose mothers were heavier drinkers during pregnancy show clear performance decrements in tests requiring vigilance and fast reaction. This suggests that although the children of the heavier-drinking mothers may be clinically normal, their lowered vigilance and longer reaction time may represent minor alcohol-related CNS perturbation along the same continuum that includes the severe neurological and behavioral deficits that occur only at alcoholic levels of consumption among mothers, that is, in FAS.

Mechanisms of Effects on the Fetus

Research on the possible teratogenic mechanism involved in FAS and ARBD has centered around direct alcohol toxicity, placental dysfunction, nutritional deficits, fetal hypoxia, and acetaldehyde toxicity. For a review of this research, see Randall (*29*). This paper briefly examines several potential mechanisms of certain ARBD that have been explored recently.

Because CNS deficits are such an important and tragic aspect of FAS, considerable research has addressed potential mechanisms of CNS-derived injury. One brain structure that has been a focus of attention is the hippocampus. The importance of the hippocampus in learning (*30,31*), a behavior manifestly affected in FAS, contributed to the attention given to the potential fetal alcohol-induced injury of this structure. A number of studies have demonstrated neuroanatomical abnormalities in the hippocampus following prenatal exposure to alcohol (*32–34*). Some evidence also suggests that alcohol exposure during the period that in the rodent model is the developmental equivalent of the human third trimester of pregnancy (the first 10 days after birth of the rat pup), exerted a greater adverse impact on hippocampal development than exposure during the equivalent of the first or second trimesters (*35*).

In addition to the hippocampus, prenatal exposure to alcohol also causes profound disruptions in the development of the cerebral cortex. Growth and development of the cerebral cortex requires the generation of neurons within the germinal zones and their subsequent migration to specific anatomic sites. Miller observed that alcohol alters the period for neuronal generation and the migration of newly born neurons (*36*). Further, the number, size, and shape of neurons were altered by prenatal exposure to alcohol.

An issue with significant public health importance is whether the extent of fetal injury relates more to the absolute quantity of alcohol to which a fetus is exposed, or to the maximal concentration of alcohol attained within the fetus. This issue was approached by West and his colleagues (*37*) in a study with young rat pups. Identical total amounts of alcohol were administered to two groups of rat pups. For one group the alcohol was divided into 12 even doses distributed over the course of a day; for the other group, the same daily amount was provided in 4 doses. Therefore, the latter animals, though administered the same total amount of alcohol, attained higher blood alcohol concentrations. The animals receiving alcohol in 4 doses had smaller brains for body size (microcephaly) than either the animals receiving alcohol in 12 doses or control animals receiving no alcohol. These decrements persisted past age 90 days. Further deficits in balance and hindlimb coordination were observed. That study indicated (a) the importance of alcohol concentration in relation to alcohol dose, and (b) exposure to alcohol in the third trimester results in persistent injury to the CNS.

An important finding arising from research is that prostaglandins may be involved as a mechanism of alcohol-induced birth defects. Prostaglandins are a class of lipids that exert hormone-like activities. Randall and Anton have observed that aspirin, a drug that functions as an inhibitor of prostaglandin synthesis and that can cross the placental membrane, antagonizes the teratogenic effects of alcohol (*38*). Though aspirin did not totally eliminate pathology caused by alcohol exposure, it did significantly reduce the number of fetal malformations.

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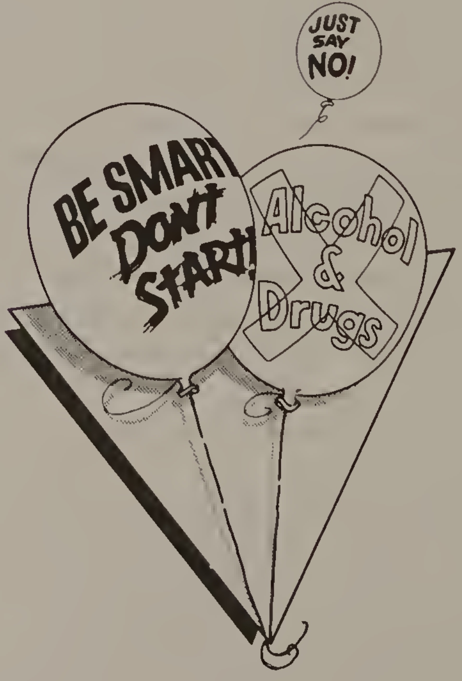
JUST SAY NO!
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Alcohol & Drugs

Children of Alcoholics: Helping a Vulnerable Group

MIGS WOODSIDE

Ms. Woodside is the Founder and President of the Children of Alcoholics Foundation, a voluntary, nonprofit organization created in 1982. Tearsheet requests to Children of Alcoholics Foundation, 31st Floor, 200 Park Ave., New York, NY 10166.

Synopsis

There are 28 million children of alcoholics in the United States—1 of every 8 Americans. They are more likely than others to suffer from alcoholism and a wide range of physical, emotional, and mental health problems. It is probable that an inherited predisposition for the disease of alcoholism exists.

Most children of alcoholics do not become alcoholic, but they are at increased risk for many other health problems. Records of the use of services provided by health maintenance organizations and of health insurance claims show that children of alcoholics use more medical and hospital services than other children. Children of alcoholics are more likely to have problems in school and to abuse alcohol and other drugs. Their mental and physical health problems persist into adulthood.

Clinical findings show that life in an alcoholic family is often characterized by pain, guilt, fear, tension, and insecurity. Children do not know that alcoholism is a disease which they cannot cause, control, or cure. Because alcoholism is a family secret, children rarely seek help, even as adults.

Because the children of alcoholics are in many medical and social service systems, greater awareness and understanding by health and human service professionals can lead to identification and help for this vulnerable group. It is critical for family physicians, obstetricians, pediatricians, nurses, social workers, hospital staff, and others to incorporate questions about family alcoholism in routine screening procedures for youth and adults. Recommendations and useful materials are discussed.

THE EXTENT of our nation's alcoholism problem is widely known and documented. However, only within recent years has knowledge about the effects of parental alcohol abuse on children been developed. We now know there exists a large but hidden group of youngsters and adults who are especially vulnerable to the disease of alcoholism and to a wide range of alcohol-related physical, emotional, and mental health problems. These are the children who grow up in alcoholic families. They are truly children at risk.

There are 28 million children of alcoholics in the United States—1 of every 8 Americans (*1*). Seven million are youngsters under age 18 who face the fear, uncertainties, and problems which result from parental alcohol abuse. Twenty-one million are adults who may continue to suffer long-lasting problems caused by life with an alcoholic parent. Despite their numbers and their overrepresentation in service delivery systems, children of alcoholics are rarely identified or helped. Yet, professionals in health and social service systems are in prime positions to provide assistance. Millions of children from alcoholic families can be helped if physicians, social workers, therapists, mental health specialists, counselors, and educators understand their risks and needs.

Research on Children of Alcoholics

Research has shown that alcoholism runs in families. Although no biological marker or specific DNA has yet been identified to predict familial alcoholism, it is probable an inherited predisposition for the disease exists. Sons of alcoholic fathers are four times more likely than others to become alcoholics (*2*). Daughters of alcoholic mothers are three times more likely to become alcoholic than other daughters (*3*), and they are more likely to marry alcoholic men, perpetuating the cycle of family pain on future generations (*4*).

Yet, most children of alcoholics do not become alcoholic. Many appear to lead successful lives. But as a group they are at greater risk than others for a

'. . . children know instinctively that they cannot discuss their parent's drinking with outsiders. They are often terrified that someone will notice or mention what everyone knows, that the parent drinks too much. For these reasons, children of alcoholics have been silent.'

variety of mental, physical, and emotional problems (*5*). Some children, whose mothers abused alcohol during pregnancy, are born with Fetal Alcohol Syndrome (FAS), the leading known cause of mental retardation in the Western Hemisphere. A recent review of the literature confirms the likelihood of multiple cases of FAS in families. If one child in a family is born with FAS, there is an increased probability that other children, especially younger siblings, will also be born with FAS (*6*).

In infancy, children of alcoholics are more likely than other children to have feeding problems and be prone to vomiting and incessant crying. As youngsters, they are more likely to have headaches, abdominal pain, tiredness, sleep problems, tics, nausea, and enuresis for which no physical causes can be found (*7*). Children of alcoholics are more prone to migraine and asthma (*8*), and to have allergies, anemia, frequent colds or coughs, and weight problems (*9*).

Children of alcoholics tend to use medical and hospital services more often than others. A comparison of the use of services of a health maintenance organization by children of alcoholics, from preschool through teen ages, with other youngsters showed the sons of alcoholics had 60 percent more injuries, were five times more likely to report emotional problems, and were two and one-half times more likely to be classified as severely ill or disabled (*10*). In the HMO study, hospitalization rates for daughters of alcoholics were three and one-half times higher than that of others, and their likelihood of attending a counseling session was three times higher than it was for other daughters. Preschool children of alcoholics, regardless of gender, were 65 percent more likely to experience an illness than other youngsters. The youngest daughters and eldest sons of alcoholic parents were those children of alcoholics at highest risk for serious illness.

Similar findings have been reported by other researchers. In one investigation 4- to 15-year-old children of alcoholics had 8 percent more medical visits and visits for treatment, more than double the number of injuries needing treatment, and five times more psychiatric examinations than other children (*11*). Numbers of hospital admissions, dispensary care visits, and psychiatric examinations were all higher for children of alcoholics.

However, large-scale studies of health problems of children of alcoholics do not exist. While exhaustive data have been developed on the health care costs of alcoholics and their families, data are not reported by children's age, sex, or type of problem. To generate this crucial information for use by health and social service systems, the Children of Alcoholics Foundation and the Philadelphia Health Management Corporation are carrying out an investigation based on 2 million health insurance subscribers to Independence Blue Cross. Preliminary results from this study provide new evidence of the impact of alcoholism on the medical and mental health of children. Compared with others, children of alcoholics (through age 19) are more vulnerable to both general and specific health problems. They have higher admission rates for hospital care and spend more days in the hospital—an average of 7.6 days compared with 5.9 days for children from nonalcoholic families. As a result, the costs of their health care are higher (*12*). By the time children of alcoholics reach school age, cognitive and behavioral problems may be manifest. One study, which detected poorer performance skills as early as 4 years of age, also noted that children of alcoholics had lower academic levels and did not perform as well as others in abstract problem solving (*13*). A comparison of 40 elementary school-aged children of alcoholic mothers with other youngsters showed the children of alcoholics had significantly lower test results in mathematics, reading recognition, and reading comprehension. They were also more likely to be assigned to special education classes (*14*).

The negative effects of parental alcohol abuse often persist in adolescence. A prospective study of young men at high risk for alcoholism reported that as students they were more likely to be involved in fights in school, and they have more reading difficulties and other academic problems for which they were referred to school psychologists. They also attended significantly more schools than others and reported their childhoods as having been less happy and their home conditions as more unstable (*15*). In a 20-year investigation of 259 multiproblem children, which included 144 children of alcoholics, only 45 percent of those from alcoholic families finished high school. Reasons for leaving included expulsion, early marriage, pregnancy, military enlistment, and institutionalization. Difficulties were noted as early as the elementary school years. Counselors detected psychological problems for 15 percent of the group during junior high and 5 percent of the group during elementary school years (*16*).

Research on substance abuse by adolescent children of alcoholic fathers indicates they are more likely to have tried marijuana, hashish, speed, and cocaine than youngsters with nonalcoholic or depressed fathers (*17*). A significant relationship has been found between alcohol abuse by teenage boys and alcoholism in their biological mothers and fathers (*18*). Preliminary research also points to a relationship between adolescent suicide attempts and parental alcohol abuse (*19*).

The problems of childhood and adolescence also carry over into adulthood. Adult sons of alcoholics make more visits to hospitals, receive more surgical and ambulatory care, and make more visits to physicians for drug and alcohol abuse than others. Daughters of alcoholics receive more gynecological services (*8*).

In a study of men in treatment for alcoholism, those with alcoholic first-degree relatives had more depression, panic attacks, obsessive-compulsive disorders, anger and hostility, more drug abuse, sleep disturbance, severe nightmares, and memory impairment than other alcoholics (*20*). In addition, they were more likely to have been hospitalized, arrested, fired, or separated from a loved one due to drinking. The age of onset of their alcoholism was earlier than that for other alcoholics, and the disease was more severe.

Likewise, women from alcoholic families show an earlier onset of problem drinking. A study of college-aged daughters of alcoholic fathers compared with daughters of nonalcoholics showed they had different drinking patterns as early as their late teens, including greater daily consumption as well as more drinking on separate occasions. They also had more drinking-related problems with friends and more drinking as a coping method when faced with personal problems (*21*). A study of bulimic women and their families found that alcoholism was the most frequently occurring psychopathology in their first- and second-degree relatives (*22*).

It is important to emphasize that not all children from alcoholic families become alcoholic, nor suffer mental, emotional, and physical problems. There is evidence that some children may be more resilient than others; they appear to do well despite their home environment. Much more needs to be learned about the levels of risks as well as the protective factors that may affect a child's vulnerability to familial alcoholism and its related problems. Increased research in this area could be extremely valuable in learning why some children of alcoholics are affected to a greater degree than others.

'The majority of survey participants believed that children of alcoholics were more likely than other employees to have low self-esteem, use drugs or abuse alcohol, have a lot of health problems, and be depressed. Although they agreed that parental alcohol abuse has a carryover effect, few recognized that these effects could result in problems in the workplace.'

Life in an Alcoholic Family

Studies in biogenetic and psychosocial research are essential, but they cannot capture the emotional quality of life in an alcoholic family. Clinical findings and case narratives detail the pain, guilt, and loneliness that parents' drinking causes children (*23*). Youngsters truly believe that they can cause their parents to drink. They do not know or believe that alcoholism is a disease. They are convinced that if they got straight ''As'' in school, remembered to make their bed, or did not use the phone, their parent would not drink. Parents often blame children for their drinking, and the resulting guilt often leads the children on a hopeless quest for perfection.

Because the alcoholic parent is the center of the family's attention, the child often feels unwanted, unloved, unimportant, and invisible. Home life is characterized by tension and insecurity. An alcoholic parent may be effusively affectionate in the morning and physically abusive at night, depending on the level of alcohol in the bloodstream. Children never know what to expect. To survive, they learn to read signals to find out if it is safe to ask a parent for money for a movie or for help with homework. They know that their simplest request can trigger a family maelstrom.

Children of alcoholics are frequently isolated. Bringing home a friend is risky because of the embarrassment or humiliation if the parent is drunk. Children miss out on the social relationships vital to their ability to function later as adults. Alcoholic delusions are especially painful for children. They do not understand what is happening when a parent talks to imaginary companions or acts as though the child wasn't there. During these episodes, children cannot determine what is real or unreal. They often think something is wrong with them and that they are going crazy. Children do not know what is normal.

Ways to Help

While researchers continue to explore the genetic, biomedical, and psychosocial factors in the transmission and effects of family alcoholism, much can be done through education, identification, and early intervention to break the cycle and alleviate the pain and suffering for many. Because children of alcoholics exist in all medical and social systems, greater awareness and understanding by health and human service professionals can lead to identification and help for this vulnerable group.

However, until recently, three factors conspired to keep children of alcoholics hidden and invisible. First, denial is inherent in the disease of alcoholism. Usually, alcoholic parents deny that they have a drinking problem and commonly believe, ''I can quit—anytime.'' Most often, their sober spouse and the children are afraid to discuss the drinking. The entire family lives with, but denies, that alcohol is ruling and ruining their lives. Secondly, children know instinctively that they cannot discuss their parent's drinking with outsiders. They are often terrified that someone will notice or mention what everyone knows, that the parent drinks too much. For these reasons, children of alcoholics have been silent. They have no recourse to sympathetic physicians, nurses, or teachers. They learn early, almost by osmosis, that alcoholism is a family secret.

Third, in our society, alcoholism carries a social stigma. Even as adults, children of alcoholics generally do not acknowledge their parent's alcoholism. They still harbor feelings of shame and may believe that talking about a parent's alcoholism constitutes betrayal, even when the parent has moved away or is deceased.

Entrenched patterns of denial prevent them from realizing the damage that their parent's drinking may have caused. Now, spurred by tremendous public and media attention to this issue, children of alcoholics are at last coming forward to ask for help.

Unfortunately, help is not always available. Because little is taught in medical or professional schools about the long-lasting, harmful effects of parental alcohol abuse, health and human service practitioners may not have the knowledge nor tools they need to respond. Yet it is critical for family physicians, obstetricians, pediatricians, nurses, social workers, and hospital staff, among many others, to incorporate questions about family alcoholism in routine screening for both youth and adults. Professionals need to be alert to indications of family alcoholism. Broken bones, lacerations, and child abuse may well indicate parental alcoholism. Suicide attempts by children and adolescents can be triggered by parental drinking. Special attention should be paid to children or adults who have these symptoms:

- somatic problems, especially headache, fatigue, abdominal pains, and sleep problems;
- a persistent inability to cope with life problems, seek or accept help, trust others, and foster satisfying intimate relationships;
- eating disorders, particularly bulimia; and
- drug or alcohol abuse problems.

The principal goal of the care provider is to help those who grow up in an alcoholic family to identify the problems caused by parental alcoholism and to guide them to appropriate care. Because children of alcoholics usually deny their parent's alcoholism, service providers need to question and probe gently in an atmosphere of trust and total confidentiality. In general, children of alcoholics experience both guilt and tremendous relief when the secret is exposed. Professionals need to underscore that alcoholism is a disease and that children of alcoholics are more vulnerable than others to the illness; therefore they need to know what steps they should take to reduce the child's risks.

In some communities there are alcoholism treatment personnel who specialize in talking to young and adult children of alcoholics. Some mental health clinicians also have had sufficient experience with alcoholism to be good resources. Health care providers can assist patients in making contact with alcohol programs and Al-Anon and Alateen self-help groups.

Sometimes supplying a name and phone number may not be enough. In general, it is advisable for practitioners to arrange a return visit or followup phone call to help the patient take action. Health and human service professionals should also be prepared to suggest books and other materials as well as the local resources for people who may need long-term group or individual therapy. Therapists, psychologists, psychiatrists, and mental health specialists need to know about the dynamics of family alcoholism and to ask questions. Too often, children are treated for depression when the major source of their problems is a parent's drinking. When professionals know about children of alcoholics' issues, the course of therapy can often be much less protracted. These and other suggestions are contained in the Health Education Packet developed by the Children of Alcoholics Foundation. These materials were the first targeted especially to health care providers and their patients. The information has helped health care practitioners to identify and appropriately assist children from alcoholic families. In answering evaluation questionnaires, a high percentage of respondents said that the materials provided them with new knowledge and that they planned to work with their patients to raise and deal with issues that stem from parental alcoholism.

Presently, the Foundation is developing the first medical education program for practicing physicians, medical schools, and health care institutions. The program, "Children of Alcoholics: A Population at Risk," draws heavily on research studies as well as the Foundation's national collection of more than 500 drawings and pictures by children of alcoholics. The program also includes a self-instructional manual and three video cassettes to improve physician-patient interviewing skills through role modeling.

At this time, there are relatively little workplace data on adult, employed children of alcoholics, particularly information about their performance on the job or their use of employee assistance programs. In 1986, the Children of Alcoholics Foundation, with the firm of Yankelovich, Skelly, White/Clancy & Shulman surveyed 36 companies nationwide to learn what corporate medical directors and employee assistance personnel know about adult employed children of alcoholics (*24*). The majority of survey participants believed that children of alcoholics were more likely than other employees to have low self-esteem, use drugs or abuse alcohol, have a lot of health problems, and be depressed. Although they agreed that parental alcohol abuse has a carryover effect, a few recognized that these effects could result in problems in the workplace. Health care and human services professionals working in employee health programs need to become better informed and educated about these problems as a work force issue and to develop well-conceived and well-planned programs to help employees realize their full potential.

There is no need to create new bureaucracies and new systems to deal with children from alcoholic families. What we do need is to educate health care and other professionals who are in daily contact with children of alcoholics about this major social and public health issue and give them effective ways to help this huge and greatly neglected vulnerable population.

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The Role of Alcohol in Suicides in Erie County, NY, 1972–84

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Synopsis

A discriminant analysis of the 806 suicide victims in Erie County, NY, from 1972-84, compared those with alcohol in the blood to those without. Thirty-three percent of the victims had alcohol in their blood. Those with blood alcohol present were more likely to demonstrate such characteristics as being male, leaving no note, being found in a vehicle, having no prior attempt, using a gun, killing themselves in the evening or at night, and not being under psychiatric treatment.

The results are interpreted to mean that alcohol is a contributory cause of impulsive suicides. Suicides related to long-standing conditions, such as chronic depression or physical illness, which are less spontaneous and more predictable, are less likely to involve alcohol. The alcohol-related suicide is more likely to be impulsive.

Alcohol-involved suicides reflect general drinking patterns, with men drinking more than women, and most drinking being done in the evening.

THE SUICIDE RATE in the United States has been increasing since the late 1950s (*1,2*), although a recent, slight decrease has been noted (*3*). The suicide rate for males notably exceeds that for females. Suicide rates are highest among elderly males, although recently their rate has decreased (*1,2*). The suicide rate for whites usually is greater than that for most other races, with the exception of American Indians (*2*). Suicide is less prevalent in Midwestern States and more common in Eastern and Western locales (*2*). Self-inflicted death tends to occur at a higher rate in urban areas (*4*).

Adolescents show the greatest increase in suicide rates, a change especially noticeable for males (*1,2,5*). Self-inflicted gunshot wounds, followed by poisoning and asphyxiation, are the most common methods of suicide (*2*). Boyd (*1*) concludes that the rise in suicide by firearms drives the overall increase in the suicide rate. Brent and coworkers (*5*) have found that suicide by firearms has increased faster than all other methods in their study of youthful victims in Allegheny County, Pennsylvania, during 1960–83.

Alcohol is frequently found in the blood of suicide victims. Roizen (*6*) found that 15 out of 20 studies of suicide had shown alcohol in the blood of 20 percent or more of the victims. Haberman and Baden (*7*) found alcohol in the blood of 32 percent of suicide victims in New York City. Ford and coworkers (*4*) found alcohol in the blood of 25 percent of those who committed suicide in Cuyahoga County (Cleveland), Ohio, during 1959–74.

Not only is alcohol often found in the context of suicide, but suicide is an unusually common cause of death among chronic heavy drinkers (*8*). However, the nature of the relationship between alcohol and suicide is not clear. The acute influence of alcohol may contribute to suicide for some people who otherwise would not have done so.

Russell and Mehrabian (*9*) recognized the importance of situational variables—the setting, prior emotional state, personality, and alcohol dose—on the drinker's emotional state. The acute effects of moderate alcohol intake may lead to disinhibition and therefore increased risk taking, anger, and a higher level of anxiety, especially in stressful situations (*9*). Unpredictable, explosive, and aggressive behaviors characterize the "abreactive" suicide attempts that are associated with the onset of a drinking episode (*10*).

Except for large doses, which tend to exacerbate depression, alcohol can either increase or decrease depression depending on the original emotional state of the drinker (*9*). Mayfield and Montgomery (*10*) found a depressive type of suicide attempt that tends to follow a prolonged bout (greater than 2 weeks) of excessive drinking.

It is likely that the deterioration of lives caused by chronic alcoholism contributes to the despair that leads to suicide. Kendall (*11*) found that suicide tends to come late in the cycle of alcoholism, usually preceded by such high risk factors as attempted suicide and divorce.

Alternatively, there may be no causal relationship between alcohol and suicide, except that the types of persons who drink frequently are more likely to kill themselves. However, Bell and coworkers (*12*), in a study of the general population, found no association of suicide ideation and high depression with drinking patterns.

Examination of the circumstances of alcohol being present in the blood of suicide victims helps explain alcohol's role in suicide, about which little is known. For this reason, we systematically compared suicides in which alcohol was present to those in which it was not.

Methods

Erie County is in the northwestern part of New York State. Erie is the 24th largest county in the United States, with a population of about 1,015,500. Buffalo is the main city in the county, with a population of 357,900 (*13*). The 1980 census (*13*) found that the population of Erie County was about 88.1 percent white, 10.2 percent black, 1.4 percent Hispanic, .6 percent Asian, and .5 percent American Indian.

Table 1. Alcohol in the blood of suicide victims by sex, race, and age; Erie County, NY, 1972–84

Characteristic	*Victims with alcohol in blood*	
	Number	*Percent*
Male	552	37
Female	254	26
White	720	34
Black	63	33
Hispanic	8	38
American Indian	13	39
Younger than 21 years	101	30
21–40 years	353	35
41–60 years	248	38
Older than 60 years	104	24

NOTE: Chi-square statistic for sex is significant at .01 level. Chi-square statistic is not significant for race and age at .05 level.

Information concerning all suicides in Erie County, in the period 1972–84 was obtained from a retrospective analysis of the files of the Chief Medical Examiner. The files contain a standard "Identification of Body Form" questionnaire which describes the race, sex, age, and date of death of the victim; circumstances of the death; and results of the toxicologic analyses of blood alcohol level (BAL) and drug content. All suicide cases in the files were included in the study. Sampling of BAL usually was performed within 8 hours of death. Gas chromatography was used to measure blood alcohol content.

The medical examiner estimated the time of death, which might differ from the time that the body was found. Interviews with the physician and relatives of the victim and a search of hospital records provided information on past psychiatric treatment. The apparent reason for the suicide was determined from a note, if one was left, and through interviews with the victim's friends and family. Interviews and record searches, as well as physical evidence, such as scars, were used to determine any history of suicide attempt. Our analysis strategy was:

(*a*) A stepwise discriminant function analysis was used to select variables able to discriminate with statistical significance ($P<.1$) between suicide victims with alcohol in the blood and those without. We used the liberal $P<.1$ criteria in examining a range of variables which could still be interpreted in light of their statistical significance, including age, race, sex, time of day of the suicide, history of suicide attempt, presence of a suicide note, history

Table 2. Discriminant analysis of presence of group variable and the absence of alcohol in the blood of 806 suicide victims

Predictor	Standardized coefficient	Significance of F-to-remove	Interpretation
Tranquilizer in blood	.31	.01	Tranquilizer in blood, HPA [1]
Sex	.24	.07	Males, HPA
Note	−.24	.05	Left no note, HPA
Age in years [2]	.25	.04	Age 21–60, HPA
Human relations problem	.33	.01	Human relations problem, HPA
Found in vehicle	.45	.001	Found in vehicle, HPA
Use of gun	.24	.06	Used gun, HPA
Prior attempt	−.22	.09	No prior attempt, HPA
Evening or night	.37	.002	Died in evening or night, HPA
Psychiatric treatment	−.22	.10	No psychiatric treatment, HPA

[1] HPA = higher probability of alcohol in blood.

[2] 21 through 60 years versus younger than 21 or older than 60 years.

Table 3. Suicide predictability score and alcohol in blood of 806 suicide victims

Score	Number of suicides	Percentage with alcohol in blood
0	353	43
1	262	29
2	157	25
3	31	23
4	3	0

NOTE: Chi-square statistic is significant at .0001 level.

of psychiatric care, place where the body was found, apparent reason for the suicide, method of suicide, and drugs in the blood. Categorical variables were represented by 0, 1 "dummy" variables.

Stepwise discriminant analysis is a statistical technique that selects the variables that best show the differences between two or more groups. The stepwise technique sequentially enters variables into a discriminant function—a linear combination of variables that separates the groups—and retains those variables that meet a specific probability criterion. The predictor variables are interpreted by their standardized coefficients, which are similar to the beta coefficients used in multiple regression. Positive coefficients indicate predictor variables that are associated with large values of the discriminant function; negative coefficients are associated with small function values. In this study, large discriminant function values indicated a higher probability of alcohol in the blood of the victim, while low values indicated a lower probability.

(*b*) Pursuing effects uncovered by the discriminant analysis, we examined the relationship between "predictability" of a suicide and the presence of alcohol in the blood, as well as the relationship between chronological patterns of suicide and alcohol in the blood.

Results

The demographic characteristics of the suicide population (table 1) tend to coincide with those of the county, with a few notable exceptions. None of the suicide victims was recognized as of Asian descent. The percent of suicides by American Indians (about 1.6 percent of the total) was more than triple their proportional representation in the population of Erie County.

Thirty-three percent of the 806 suicide victims in Erie County from 1972–84 had alcohol in the blood. Table 1 shows the percent of victims with alcohol in the blood for several demographic categories. Men were significantly more likely to have blood alcohol content than women, but there was no significant relationship with race or age. Of those victims with alcohol in the blood, 86 percent had BALs greater than or equal to .05 percent. Sixty percent of the alcohol-bearing cases were legally intoxicated (BAL of .1 percent or higher).

Table 2 shows the results of the discriminant function analysis. The suicide victims most likely to have alcohol in the blood showed the following characteristics: male, aged 21–60 years, tranquilizer in the blood, no suicide note, committed suicide because of human relations problems, committed suicide in a vehicle (not auto crash), used a gun, no known suicide attempt, committed suicide in the evening or at night, and not under psychiatric treatment. The relationships with sex and age are not surprising. In the general population, males drink more than women; young adults and the middle aged drink more than children or the elderly (*14*). Drug users drink more than the average (*15*), although it is not clear why drugs, other than tranquilizers, showed no significant relationship with alcohol. There is more drinking in the evening and night than in the morning or afternoon.

Table 4. Distribution of 800 suicides over 28 weekly time slots, percent with alcohol in blood, and mean predictability scores

Characteristic	*Morning*	*Afternoon*	*Evening*	*Night*	*Total*
Monday					
Percent with alcohol	29	30	47	38	33
Number	38	47	17	16	118
Score	.58	.98	.67	.94	.80
Tuesday					
Percent with alcohol	52	16	37	49	34
Number	21	43	38	23	125
Score	.52	1.02	.89	.75	.85
Wednesday					
Percent with alcohol	21	20	30	36	25
Number	28	30	37	11	106
Score	1.00	.93	.89	1.09	.95
Thursday					
Percent with alcohol	32	41	43	42	39
Number	31	44	28	12	115
Score	.61	.91	.72	.83	.78
Friday					
Percent with alcohol	33	31	43	43	35
Number	36	39	28	7	110
Score	1.03	.98	.71	.57	.90
Saturday					
Percent with alcohol	29	25	29	62	34
Number	31	28	31	21	111
Score	1.00	.93	.68	.86	.87
Sunday					
Percent with alcohol	34	24	42	48	36
Number	29	34	31	21	115
Score	.90	1.00	.62	.62	.80
Total					
Percent with alcohol	32	27	38	47	34
Number	214	265	210	111	800
Score	.81	.97	.76	.80	.85

The analysis of significant variables suggests that alcohol-related suicides are more spontaneous and less predictable than nonalcohol-related suicides. To test this possibility, we constructed a "predictability" scale. Suicides were given one point for each of the following factors: left a note, diagnosed as depressed, poor health as the cause, suicide attempt, and under psychiatric care. This scale reflects the extent to which a suicide is related to a long-standing condition (suicide attempt, depression, psychiatric care, or poor health), or is not impulsive, as suggested by some preparation. Use of a gun could be associated, with impulsive suicide for victims who already had a gun. However, the connection between use of a gun and spontaneity was not clear enough to include in the scale.

Table 3 shows the relationship between the scale and alcohol in the blood of the victim. Suicides which were less predictable and more impulsive were more likely to show alcohol in the blood of the victim.

To examine a chronological factor for suicide in Erie County, we created a collapsed data set of 28 cases corresponding to 28 6-hour time periods in the week (table 4). The three variables of interest were the number of suicides during the "time slot," the percent of victims with alcohol in the blood, and the mean "predictability" score for suicides in that time slot. Table 4 shows that a larger proportion of suicides that occur in the evening and night involve alcohol than those that occur in the morning and afternoon. Suicides in the evening and night tend to have low mean "predictability" scores.

Table 5. Correlation matrix of suicides over 28 weekly time slots by percent with alcohol in blood and mean predictability scores

Variables	*Percent with alcohol*	*Number*	*Score*
Percent with alcohol	1.00	. . .	. . .
Number of suicides	[1] −.52	1.00	. . .
Mean predictability	[2] −.49	[3].31	1.00

[1] P = .002. [2] P = .004. [3] P = .052.

We correlated the three variables used in the time slot analysis (table 5). As noted, suicides with alcohol have lower "predictability" scores. They tend to take place at night when the number of suicides generally is lower. There is a positive relationship between the predictability of suicides and their occurrence when suicides are more common. The picture emerges of a certain type of suicide, characterized by unpredictability, with alcohol in the blood, and happening at night.

Discussion

Our data suggest that alcohol tends to be associated with those suicides that are impulsive, rather than those based on long-standing deliberation. There is more likelihood in the alcohol-related suicide for there to be no note, no known suicide attempt, and no long-standing condition to which the suicide can be related. The weapon is more likely to be a gun. The victim is more likely to be found in a vehicle. Our characterization of the alcohol-related suicide is congruent with the findings of Peterson and associates (*16*), who studied 30 cases of nonfatal, self-inflicted gun-shot wounds. They found that 15 had used alcohol or drugs before the wounding, that 18 had experienced interpersonal conflict, and that none had written a suicide note. They hypothesized a special type of impulsive, gun-wielding suicide attempter.

Our findings that alcohol-related suicides are more prevalent at night agree with the results found by Varadaraj and Mendonca (*17*) in their analysis of 158 suicide attempters admitted to an emergency ward at a British hospital. Higher blood alcohol levels were associated with male suicide attempts on weekends and with female attempts on weekdays; females had significantly higher BALs during nighttime attempts (*17*).

Conclusion

Does alcohol play a causal role in any of these suicides? Unlike homicide, suicide is least prevalent during the peak evening drinking hours. It is possible that most victims simply drink to prepare themselves for what they already are determined to do. However, if this were so, they might be expected to leave notes or give other signs of preparation. It is possible that the type of person who commits suicide impulsively is more likely to be a drinker.

Nevertheless, the large number of persons under the influence of alcohol who kill themselves (*5,6,7*), and the correlation that we found of drinking with impulsive suicide, strongly suggest that significant numbers of suicide victims would not have committed the act had they not been drinking. Alcohol clearly is implicated as a causal factor and a frequent triggering agent in suicide.

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Alcohol and Highway Safety in a Public Health Perspective

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Synopsis

The Public Health Service and the National Highway Traffic Safety Administration share the responsibility for problems related to injury prevention and control regarding the alcohol-impaired operation of motor vehicles. NHTSA activities have evolved over several decades within a general framework which emphasizes community-based systems.

The National Highway Traffic Safety Administration is promoting program activities that stress community-level involvement in problems of alcohol and highway use. The public health approach to the mortality and morbidity resulting from alcohol use and motor vehicle operation entails examining and promoting those activities that address human factors. Techniques for Effective Alcohol Management (TEAM) is a cooperative effort representing sports, entertainment, insurance, vehicle manufacturer, and other organizations and agencies building community coalitions. The Centers for Disease Control is establishing research and collaborating centers to stimulate studies and exchange information on injury-related research. Alcohol countermeasures programs include training for law enforcement and legal officials, technology development efforts, and changes in laws applied to use of alcohol and other drugs. Outreach and networking activities have encouraged the initiation and coordination of community level groups active in promoting highway safety with regard to the use of alcohol.

Statistical method changes are being discussed for surveillance of motor vehicle-related injuries for Health Objectives for the Nation for the Year 2000. NHTSA data systems being discussed are thought to be more timely and more sensitive to crash activity than methods now in use.

Public health approaches to the problems of alcohol and highway safety are benefiting from growing cooperation among highway safety and public health officials to reduce the morbidity and mortality resulting from operation of motor vehicles by performance-impaired drivers.

THE PUBLIC HEALTH APPROACH to the prevention and control of injuries from alcohol related highway accidents focuses on three major elements. They are the environment, including such factors as alcohol availability and road design; the host, namely drivers, passengers, pedestrians, or anyone subject to vehicular injuries; and the agent, which is the energy of the injury causing collision. Programs using this approach have shifted from the Department of Health, Education, and Welfare (DHEW) to the Department of Transportation (DOT) for the past 30 years. Only now, with the creation of the Centers for Disease Control's (CDC) Center for Environmental Health and Injury Control (CEHIC), do we see both agencies actively working to solve the problems of highway safety.

Major attention to the issues of highway injury and safety was a responsibility of the Public Health Service (PHS) of DHEW, now the Department of Health and Human Services (DHHS). Then, the Federal Government's programmatic response to vehicular safety was in the transportation field, first at the Department of Commerce's Bureau of Public Roads, and after the creation of the National Highway Safety Bureau in 1966 (*1*), with DOT.

At DOT, the major focus was on highway design and engineering, vehicle safety, and law enforcement, with secondary emphasis on behavioral interventions. Programs were the responsibility of the Federal Highway Administration, and the National Highway Safety Bureau, now the National Highway Traffic Safety Administration (NHTSA).

The organization which became the CEHIC, in August 1987, received its funding from Congress through NHTSA in order to give injury prevention

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issues new interdisciplinary and interagency attention. CEHIC manages basic research activities with a behavioral and biomedical focus, as well as State and community coordinating activities which receive grant support and assistance. NHTSA continues its applied research, data collection, and program development and management activities (*2,3*).

DHEW established a Motor Vehicle Injury Prevention Program within the Public Health Service's Disease Control Programs area in the early 1960s. A landmark report projected the costs and benefits through 1968 expected to obtain from the Federal commitment to the problem of highway crashes (*4*):

> " . . . (m)otor vehicle accidents (are) examined exclusively in terms of public health concerns. This mandate focused on the role of human factors in vehicular accidents and the amelioration of injury caused by vehicular accidents. In adopting this posture, three major factors in the vehicular accident complex—law enforcement, road design and traffic engineering—were, for the most part, excluded. This constraint had the effect of limiting the problem to considerations traditionally within the purview of DHEW, while excluding those elements which are traditionally handled by the Department of Commerce and other Government agencies."

Despite the lack of agreement about what amount of drinking may be "safe," alcohol use has always been identified as a problem in highway safety (*5–11*). By 1966, with the creation of DOT, most of the activities and responsibilities of the DHEW safety effort found a new home in the National Highway Safety Bureau, later to become NHTSA.

In the health community, the problem of alcoholism and alcohol abuse was addressed primarily by the treatment and prevention demonstration programs of PHS's National Institute on Alcohol Abuse and Alcoholism (NIAAA), in existence by 1971. However, the community-based Alcohol Safety Action Projects (ASAP) of NHTSA brought the issue of alcohol abuse, in the context of the alcohol-impaired driving problem, before the public. Each ASAP reflected the particular needs and interests of specific local sponsoring agencies. States and communities were largely responsible for their implementation. ASAP addressed at least one aspect of the motor vehicle problem not found in the original DHEW motor vehicle injury prevention program—law enforcement and the entire legal system.

By the early 1980s, ASAP field experiments were coming to an end, and NIAAA was shifting away from directly funded services. Most treatment and prevention demonstration programs were being completed. The formula grant program assisting State-sponsored alcoholism services was redefined as block grants to the States, combining previously earmarked alcohol services funds with those for drug abuse and mental health (ADM). A State could allocate the block granted funds for any mix of ADM services based on its particular population needs.

In 1980, DHHS published specific health and safety goals for the Nation to be met by the year 1990 (*12*). Accident prevention and injury control received significant attention; most prominent in this section of the report was a discussion of the role of motor vehicle accidents in unintentional injury and trauma, with alcohol use accounting for nearly 50 percent of all fatal injuries. The health prevention and promotion measures suggested as program strategies were education and information, technology, legislation and regulation, and economic incentives. Interventions addressing the dangers of alcohol use combined with motor vehicle operation were discussed and cross referenced to the section on the misuse of drugs and alcohol. DHHS attention was being revitalized with regard to injury control, and especially motor vehicle related injuries. Additional sources of information are listed in the box.

The Current Situation

Since the mid 1980s, NHTSA has been promoting program activities that stress community-level involvement in the problems of alcohol and drug-impaired drivers, including motorcycle and bicycle riders and pedestrians. NHTSA, working through its regional offices, actively encourages and promotes State and local solutions, instead of trying solely at the Federal level to solve the problems of alcohol and drug impairment, as it did with ASAP.

NHTSA's community focus is reversing the trend of the last 20 years. Human factors, particularly attitudinal and behavioral (including health and medical) variables, are the subject of increased program attention, in addition to law enforcement, road design, and traffic engineering perspectives stressed in early DOT activity. In particular, NHTSA has developed initiatives that rely overtly upon community implementation of its strategies as well as the involvement of the public health field for successful execution.

NHTSA and Public Health Activities

TEAM. Techniques for Effective Alcohol Management began in 1985 with a series of informal discussions among representatives of the facilities management industry and the National Basketball Association about the problems of alcohol-impaired driving. A year later, a study was issued detailing the implementation and preliminary results from a seven-site pilot project (*13*). The program results from a coalition formed by NHTSA; Allstate Insurance Company; CBS, Inc., the International Association of Auditorium Managers; Major League Baseball; the Motor Vehicle Manufacturers Association; the National Basketball Association; and the National Safety Council (*14*). The program is designed to reduce injuries and fatalities resulting from alcohol or drug impaired driving following sports and entertainment events, to create a safer and more enjoyable spectator environment, and to introduce more effective crowd control techniques for arenas. The activities assist managers of sports arenas and stadia in developing policies to control the sale and consumption of alcoholic beverages and to help prevent the abuse of alcohol. Food and beverage service personnel and all staff members working in the facilities receive training in impairment prevention and intervention strategies. The sports facilities are designated as focal points of activity for community anti-impaired driving task forces and coalitions. Community-based networks reflect not only an area's highway safety interests, but involve representatives of the health and medical fields as well.

By the end of 1987, TEAM reported that community coalitions had been established in two cities (Detroit and Houston) (*15*), and four are in the initial stages of development (Atlanta, Denver, Phoenix, and Salt Lake City) (*16*). Alcoholic beverage service and sales practices have been introduced into and adopted by 44 facilities, with intervention and prevention training conducted for staff members at each site. TEAM has established a significant national and local public awareness campaign, having sports figures doing public service ads on television and radio, and in the print media (*15*).

TEAM is a diversion from traditional governmental approaches to assuring the public's health and well-being. Current plans call for it to be independently incorporated, a unique example of the energy and enthusiasm that can come from public-private sector partnerships (*16*), and a major, highly visible example of the community focus that NHTSA is encouraging.

Center for Environmental Health and Injury Control. In 1983, DOT requested that the National Academy of Sciences (NAS) examine the status of trauma in the United States. NAS published "Injury in America" in 1985 (*17*), reviewing injury as a public health problem and delineating five areas for which governmental support and intervention were appropriate: epidemiology, prevention, biomechanics, treatment (acute care), and rehabilitation. As a result of the report's findings, Congress appropriated nearly $10 million to DOT, to be used by the CDC for a 3-year pilot program (1986–88) in a newly created research coordination and support unit, the Division of Injury Epidemiology and Control (DIEC). Now organized as a research center within CDC, the Division solicited and reviewed the first round of research project and research center proposals, and has made funding decisions. Research centers are located at Harvard University School of Public Health, Wayne State University, University of Washington Harborview Medical Center, the University of North Carolina, and the Johns Hopkins University School of Hygiene and Public Health. Serving as an important focus of intellectual and scholarly exchange concerning injury-related research activities, the centers have been encouraged to provide training opportunities, particularly at the post-doctoral level, for professionals in the interdisciplinary field (2).

Recently, eight more institutions were approved as collaborating centers; they are at the University of California at Los Angeles, University of Colorado and the Colorado Department of Health, University of Texas Health Science Center, University of California at San Francisco and the Trauma Foundation, University of Minnesota, University of Alabama, the State University of New York at Buffalo, and the University of Vermont. Thirty research projects are being supported in the five

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needed areas cited in "Injury in America" (*17*).

NHTSA staff participated in research and center proposal reviews in order to assure that agency concerns for highway-related injury were well represented in the selection of projects during the initial funding cycle. Since then, more than two-thirds of the research grants funded have NHTSA staff members assigned to them in a coordination capacity, demonstrating the value of these projects to DOT operational and program development mandates.

The next round of grant solicitations will address services and research coordination activities, historically an area of concern in this field (*18, 2*). These projects are likely to reflect the community-level coordinated perspectives currently being encouraged by NHTSA's State and local program strategies. Awards are to be made to State and community (municipal and county) departments of health by the end of fiscal year 1988.

Alcohol countermeasures. Traditionally, the alcohol countermeasures program has developed and promoted training for the law enforcement, prosecutorial, and judicial communities in innovative techniques for the identification, apprehension, prosecution, conviction, and sanctioning of persons driving under the influence of alcohol. The program has, for example, sponsored development of standards for breath testing devices used to determine blood alcohol content, speed measuring devices, in-vehicle alcohol interlock mechanisms, and such strategies as standardized field sobriety test techniques, validation of detection cues for alcohol-impaired automobile and motorcycle operation, the sobriety roadblock or checkpoint, and the Mortimer-Filkins screening test for level of involvement with alcohol. The countermeasures program has encouraged States to adopt legislative and regulatory innovations that have demonstrated a positive effect, such as passage of a 21-year-old minimum drinking age law, administrative revocation of drivers' licenses for alcohol-impaired operation, fixed periods for license suspension, and administrative determination of guilt based on a given blood alcohol concentration, typically .10 percent BAC, known as "administrative per se."

Recently, NHTSA began expanding its traditionally alcohol-specific countermeasures to include identification of impaired motor vehicle operation resulting from use of substances other than alcohol. The drug recognition program is being pilot tested in four sites around the country, building upon the well-documented value of traditional enforcement techniques, especially standardized field sobriety tests, and adding physiological measures for determining impairment.

A project conducted jointly with the National Institute on Drug Abuse and the Office of Juvenile Justice and Delinquency Prevention concerns field testing instruments for drug and alcohol use screening and diagnosis among adolescents and young adults convicted of impaired driving (*19*). The effort was designed to continue to validate and determine the practicality of the use of instruments in the court setting that were originally developed for the adolescent drug abuse treatment field. The results are expected to augment, for adolescents, the Mortimer-Filkins screening instrument available for use among adults convicted of driving under the influence of alcohol.

Networking and outreach. NHTSA sponsors non-law enforcement activities that address the public health perspective. Among these, in additional to TEAM, are networking and outreach efforts in cooperation with professional health and medical organizations, such as the Association for the Advancement of Automotive Medicine (a series of specialized courses, research monographs, and jointly-sponsored conferences), and the Society of Public Health Education (a special issue of Health Education Quarterly on health promotion and impaired driving). Activities with constituent organizations, such as Mothers Against Drunk Driving, Remove Intoxicated Drivers, and the National Black Alcoholism Council–Blacks Against Drunk Driving, consist of issues workshops, strategy meetings, and mini-grants to local affiliate chapters to encourage coordination of activities with State and local offices of highway safety, health, and ADM departments. Networking activities involve the National League of Cities and the U.S. Conference of Mayors, and consist of workshops and strategy meetings which focus on prevention activities.

Other interagency activities. In addition to interagency projects described previously, NHTSA is working with such agencies as CDC, Office of Maternal and Child Health, Indian Health Service, and the National Committee of Injury Prevention and Control. Products include a handbook on injury prevention, a censuses workshop report, and agency plans for public health programs and strategies.

Each December, a time of increased alcohol- and drug-impaired driving, NHTSA sponsors National Drunk and Drugged Driving Awareness Week. States and localities plan special events and media campaigns for this week. In 1988, the Office of the Surgeon General will sponsor alcohol-impaired driving initiatives that correspond with National Drunk and Drugged Driving Awareness Week, among them a major national conference.

In summary, the focus of NHTSA's alcohol-impaired driving program has evolved from national level programs to encouraging State and local perspectives which see networking and outreach as the wave of the future. Emphasis is on general deterrence strategies, community focus, a systems approach, enhancing constituent impact, and encouraging prevention and intervention techniques.

Conclusions

In February 1988, DHHS published summaries of the Centers for Disease Control surveillance activities concerned with the 1990 health objectives for the Nation (*18*), noting the predominant role of injury in morbidity and mortality (the leading cause of death for those under the age of 55), and the lack of Federal commitment to its amelioration. The authors suggested that because of the unintentional nature of injury—"the accident"—injury prevention has not received major planning emphasis in government programs. They suggested that the lack of an integrated governmental response had hindered large-scale injury intervention and prevention. In the 1970s, for example, both DHEW and DOT had emergency medical services programs. Local projects funded by one department had little knowledge of or coordination with those of another. Overlap of planning and delivery of emergency and acute care services may have occurred in a number of communities throughout the country.

Most notable concerning the surveillance summary on motor vehicle-related injuries (*20*) was the discussion of proposed methods of measurement for the year 2000 health objectives. For the 1990 objectives, CDC used motor vehicle crash fatality data from its own data system (National Center for Health Statistics (NCHS) death certificate reports), and calculated fatality rates based on 100,000 population. NCHS data indicated a 2 percent higher fatality rate than comparable NHTSA figures for the surveillance period (1978–84). The author notes that the NHTSA data systems, the Fatal Accident Reporting System (FARS), and the National Accident Sampling System (NASS), are probably more sensitive to crash activity and more timely for surveillance purposes than the data CDC routinely used. The reason is primarily because a 30-day waiting period is required for case entry into FARS, making annual data available within 6 months of the close of the calendar year, rather than the 12 months post-crash reporting needed for enumeration by NCHS. NCHS data are then accessible 18 to 24 months after file closing. Additionally, NHTSA calculates rates with an exposure measure, not on a population base, using fatalities per 100,000 vehicle miles travelled, more in line with the public health perspective's concern with assessment of relative risk.

'Fortunately we are seeing growing cooperation among the highway safety and public health fields to reduce the morbidity and mortality resulting from operation of motor vehicles while impaired. We are witnessing the acknowledgement, after more than 30 years of experience, that no one discipline, or strategy, or agency, can on its own solve the tragic and costly problem.'

Fortunately we are seeing growing cooperation among the highway safety and public health fields to reduce the morbidity and mortality resulting from operation of motor vehicles while impaired. We are witnessing the acknowledgement, after more than 30 years of experience, that no one discipline, or strategy, or agency, can on its own solve this tragic and costly problem. Highway safety specialists welcome the participation of the public health and medical communities in the cooperative attack on deaths and injuries resulting from alcohol- and drug-impaired driving.

Cooperation among the public health, medical, and highway safety communities will facilitate more effective and quicker responses to the mandates of the Highway Safety Act of 1966. This equates to a healthier, safer America.

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Effects of Legislative Reform to Reduce Drunken Driving and Alcohol-related Traffic Fatalities

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Synopsis

From 1980 through 1985, considerable progress was made across the Nation in reducing drunken driving and fatal automobile crashes. More than 400 chapters of local citizen groups concerned with reducing drunken driving were formed. New media coverage, measured in number of stories, increased fiftyfold from 1980 to 1984. More than 500 legislative reforms were passed. All States now have adopted a legal drinking age of 21. Many also adopted criminal and administrative per se laws and instituted penalty increases for drunken driving. By 1985, the total number of fatal crashes declined to 39,168, a decrease of 6,116, or 16 percent, from the 1980 level of 45,284. Single-vehicle fatal crashes occurring at night, those most likely to involve alcohol, declined by 20 percent, with 3,674 fewer crashes in 1985 than in 1980. Among teenage drivers, declines in fatal crashes were steeper: Fatal crashes decreased 26 percent, and single-vehicle night fatal crashes were down 34 percent.

After 1984, however, the number of new citizen groups established and the number of stories appearing in the media began to decline. In 1986, after decreasing for several years, the number of fatal crashes rose 5 percent, and single-vehicle night fatal crashes rose 7 percent, up 1,060 from 1985. Among teenage drivers, the increase in single-vehicle night fatal crashes was even higher, up 17 percent. In 1987, single-vehicle night fatal crashes declined slightly but still remained higher than in 1983, 1984, or 1985. Legal changes, while helpful in reducing drunken driving crashes, may not be sufficient to achieve optimal long-term declines.

To sustain the progress of the early 1980s, we need to:

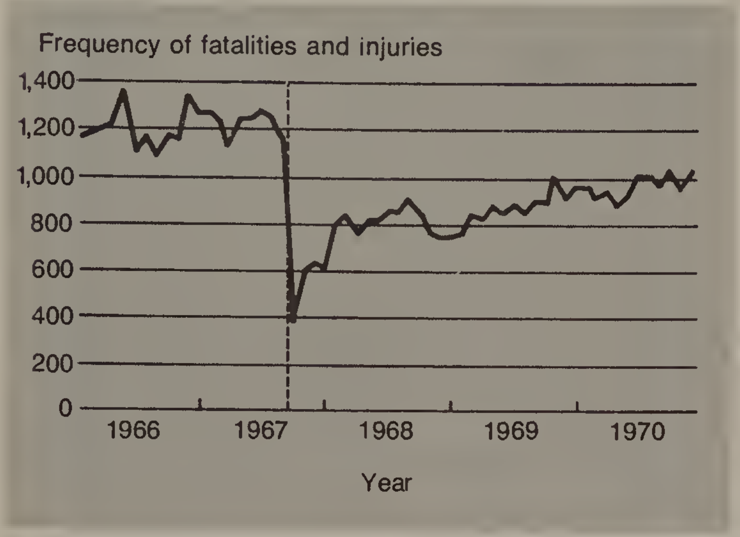
- *Refocus community and media attention on the drunken driving problem.*
- *Increase police enforcement and public support for enforcement of laws against drunken driving.*
- *Heighten educational and enforcement efforts that target risky traffic behaviors more common among drunken drivers—particularly speeding and failure to wear seatbelts.*
- *Develop educational efforts aimed at all segments of the population to increase informal social pressure to discourage drunken driving and riding in vehicles with drivers who are drunk.*

Traffic accidents remain the leading cause of death among persons ages 1 to 34; 52 percent of the 46,050 traffic fatalities in 1986 involved a driver or pedestrian who had been drinking. Without renewed attention to the problems posed by drunken driving, we may be unable to sustain the dramatic progress of the early 1980s.

A GROUNDSWELL OF PUBLIC CONCERN about the drunken driving problem occurred between 1980 and 1985, and for good reason. Traffic accidents are the leading cause of death among our Nation's youth, ages 1 to 34 (*1*). Fifty-two percent, or 23,987, of the 46,050 traffic fatalities recorded in 1986 involved a driver who had been drinking (*2*). Drunken drivers not only place themselves at greater risk of crash involvement, but 20 to 40 percent of the people killed in traffic accidents in which a driver was drunk are persons other than the drunken driver: passengers, pedestrians, or drivers of other vehicles.

During the early 1980s, citizens across the country began demanding an end to this waste of human life. From 1980 to 1985, more than 400

Figure 1. Effects of the enactment of the British Road Safety Act of 1967 on fatalities and serious injuries in the United Kingdom



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NOTE: Data combined for Friday nights, 10 pm to midnight; Saturday mornings, midnight to 4 am; Saturday nights, 10 pm to midnight; and Sunday mornings, midnight to 4 am. Corrected for weekend days per month and with seasonal variations removed.

local chapters of citizen groups such as Mothers Against Drunk Driving (MADD) were founded. News stories about the problem increased fiftyfold (*3*).

Between 1981 and 1986, 729 changes in laws were made in an effort to reduce fatal crashes involving drunken drivers (S. Hatos, U.S. Department of Transportation, personal communication, 1987). More than 40 States adopted criminal per se legislation. According to criminal per se laws, if a driver has a blood alcohol level above .10 (four to five drinks on an empty stomach), the driver is legally under the influence. Almost half of the U.S. States passed administrative per se laws. Administrative per se laws permit the drivers' licenses of drivers with blood alcohol levels above .10 to be suspended from the time of arrest until trial. In addition, many States increased the penalties for drunken driving: stiffer fines, suspension of licenses, and jail sentences for repeat offenders and for those involved in traffic accidents resulting in an injury or fatality (*4*).

From 1980 to 1985, when media attention, community organization, and legislative activity peaked nationwide, the number of fatal crashes declined 16 percent, from 45,284 in 1980 to 39,168 in 1985. Single-vehicle night fatal crashes, those most likely to involve alcohol, declined even more, down 20 percent, from 18,277 to 14,603. Among drivers in the high-risk 15- to 19-year-old group, the declines were even greater: Single-vehicle night fatal crashes declined 34 percent, with 1,285 fewer crashes occurring in 1985 than in 1980 (*5,6*).

Effects of Drunken Driving Laws

Several explanations have been offered for these dramatic reductions. First, the laws themselves may have contributed to the declines. Evaluation of drunken driving laws is constrained, however, because most States do not obtain blood alcohol information on all drivers involved in fatal crashes. Because the key dependent variable is not precisely observed, researchers have used surrogate measures, such as single-vehicle night fatal crashes. Based on estimates from States that comprehensively test alcohol levels of drivers in fatal crashes, 60 to 70 percent of single-vehicle night fatal crashes involve intoxicated drivers, compared with 33 percent of other fatal crashes.

However, single-vehicle night crashes account for less than half of the fatal traffic accidents involving intoxicated drivers, prompting some researchers to caution about the imprecision of such surrogate measures, particularly in short-term studies of small jurisdictions (*7*). Also, when several related laws are enacted within a short period of time, it is difficult to attribute effects to a single specific intervention. In addition, variables such as unemployment rates, use of seatbelts, and rates of speeding, which can all influence statistics on alcohol-related crashes, are seldom if ever controlled analytically in research evaluating drunken driving interventions.

Nonetheless, there is evidence to suggest that legal changes have produced declines in alcohol-related traffic fatalities. Well-controlled studies of increases in the legal drinking age revealed that although effects varied from State to State, in States that raised their drinking ages, night fatal crashes in targeted age groups declined 10 to 15 percent relative to States that did not (*8–11*).

An analysis of national traffic data from 1978–85 by the Insurance Institute for Highway Safety indicated that administrative per se laws, on average, reduced night crashes 9 percent relative to States that did not pass such legislation. Criminal per se laws against drunken driving and increased penalties were each accompanied by 6-percent declines in night fatal crashes relative to States that did not pass such laws. Most States have criminal per se laws against drunken driving, but only half have administrative per se laws. The Institute estimated that if all States passed such laws, 2,600 fewer fatal crashes would occur annually (*12*).

Police enforcement of drunken driving laws and court conviction rates also increased in numerous States. Quasi-experimental community studies have shown that increased police enforcement of drunken driving laws is associated with declines in crashes (*13–15*).

Social Sanctions

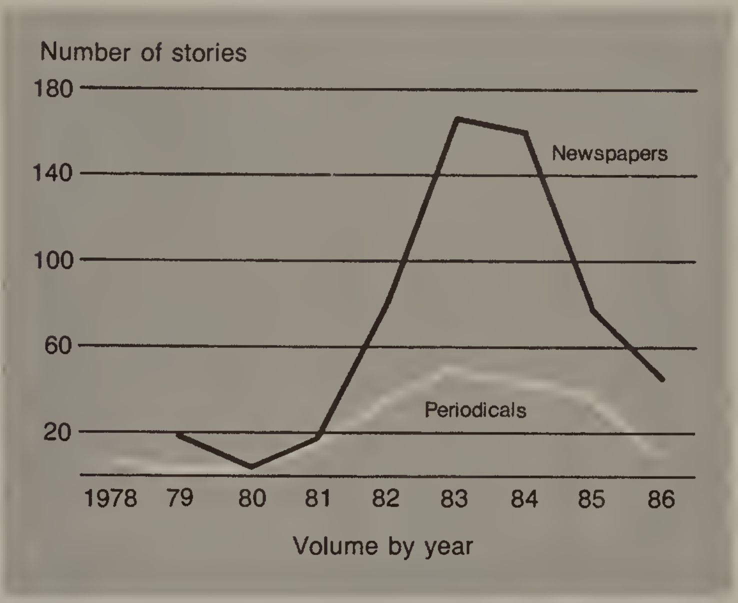
Perhaps as important as these formal legal countermeasures was the emergence of informal social pressure that discouraged drunken driving. Media attention and political lobbying often preceded new laws. Annual surveys conducted in Massachusetts from 1981 to 1985 revealed that the proportion of drivers who said they would not care at all if their best friends found out that they had been arrested for drunken driving declined from 25 percent to 12 percent (*16*). At the same time, the proportion of drivers who reported driving after five or more drinks in the preceding month declined from 18 percent to 6 percent. During that period, from 1981 to 1985, Massachusetts experienced marked fatal crash declines similar to the national trends. It should be noted that reported drunken driving and night fatal crash trends began to decline in Massachusetts and in many other States even before major legal changes occurred. Thus, the informal social pressures that stimulated the passage of drunken driving laws also may have produced reductions in drunken driving.

Warning Signs Progress May Fade

Based on the experience in Great Britain (fig. 1) and in several other countries (*1*), declines in fatal crashes after the passage of drunken driving laws can be temporary. This situation can occur if public discussion of the problem diminishes and if people become aware that the chances of being caught by the police are extremely low.

Data suggest that this temporary decline could happen in the United States as well. The number of new citizen groups formed that were concerned with drunken driving peaked in 1983 (*3*). The number of news and feature stories about drunken driving decreased sharply after 1984 (*6*) (fig. 2). After several years of decline, single-vehicle night fatal crashes rose 7 percent in 1986, while other fatal crashes rose 3 percent (fig. 3). Among teenage drivers, the increase was even greater; teenagers experienced a 17-percent increase in single-vehicle night fatal crashes, with a 6-percent increase in other fatal crashes. Our projections closely parallel U.S. Department of Transportation estimates, which show an overall increase of 5 percent in the number of drivers involved in fatal crashes who had been drinking and an increase among teenage drivers of 14 percent in 1986 compared with 1985 (*2*). Data from 1987 indicate slightly fewer single-vehicle night fatal crashes than in 1986 among all ages and among teenage drivers, but the overall total is still 5-percent higher than in 1985, and the teenage total is still 9-percent higher than in 1985.

Figure 2. Volume of national newspaper and periodical coverage of drunken driving, by year



Newspaper volume based on count of stories in the National Newspaper Index. Includes New York Times, Los Angeles Times, Wall Street Journal, and Washington Post. Periodical volume based on magazine index.

SOURCE: Reference *3*.

What Can Be Done To Sustain Declines?

It is not inevitable that the effects of drunken driving laws must fade. Several steps can be taken to maintain the national momentum in reducing drunken driving fatal crashes.

First, efforts are essential to keep the public's attention focused on this issue through local community organization and media attention. Each year, new cohorts of drivers enter the high-risk teenage driving category. It is not clear whether these new cohorts were influenced by the media attention and public discussion about drunken driving in the early 1980s. Consequently, we must continue to heighten awareness among this group. School-based education programs led by peers have been successful in reducing other health-compromising behaviors among adolescents (*17–19*) and may hold particular promise in the drunken driving area. But the programs aimed at youth must be accompanied by general programs for all age groups. Otherwise, teenagers may be given the erroneous impression that adults believe it is unsafe for teens to drink and drive, but it is an acceptable

Figure 3. Fatal single vehicle night crashes, United States, all ages, 1980–87

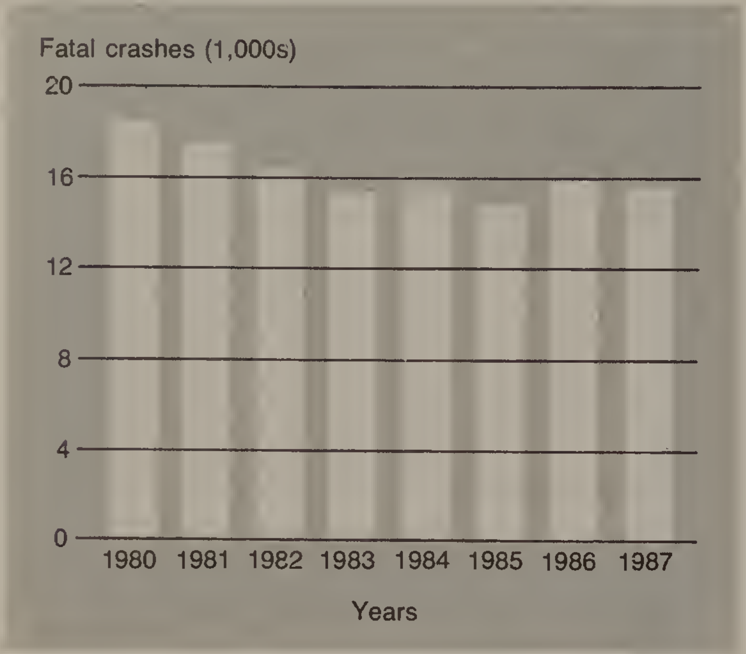
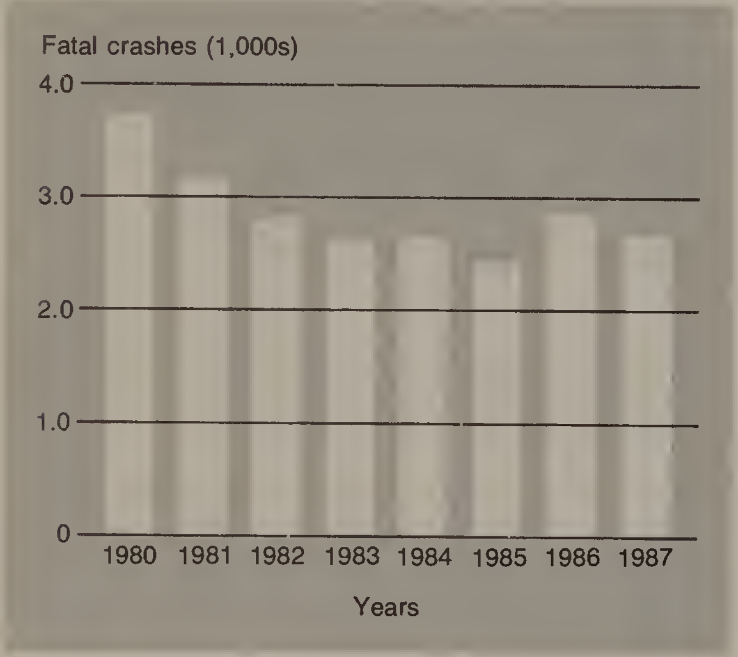


Figure 4. Fatal single vehicle night crashes, United States, teenages, 1980–87



risk for adults. Such an impression would only convey the message that drunken driving, like smoking, is a symbol of adult status.

Second, police enforcement of drunken driving laws must be increased. In recent surveys of the New England States, only one-quarter of the drivers in the sample believed it is very likely that drunken drivers will be stopped by the police, even though a majority of drivers believe arrested drunken drivers will be convicted and punished (*6*). In those New England surveys, only 3 arrests for drunken driving occurred for every 1,000 drunken driving trips reported by respondents (table 1). We must consider whether 3 arrests per 1,000 drunken driving trips can serve as a credible deterrent.

Increasing police enforcement will require both a substantial expenditure of financial resources to mobilize more officers at high-risk times and locations (namely, weekend nights on high-speed roads) and the cultivation of public support for more enforcement. Despite the low levels of arrest, a sizable minority of drivers in New England believe police enforcement of drunken driving laws is adequate. Efforts are needed to make the public aware that more enforcement can be effective (*13–15*) and that appropriately conducted, active police enforcement is a public service, not an infringement of personal liberty.

Third, we must not shift attention from other risky traffic behaviors that are more prevalent among drunken drivers. In the New England surveys, respondents who reported driving after five or more drinks in the past month, when compared with those who never reported much driving after drinking, were

- 10 times more likely to drive after marijuana and other drug use,
- twice as likely to report speeding 20 or more miles per hour over the limit and running red lights in the previous week,
- one-quarter as likely to wear seatbelts,
- four times more likely to be given a traffic ticket for violations other than drunken driving, and
- five times more likely to have been involved in a crash resulting in an injury in the past year (table 2).

An intensive analysis of Maine's 1981 comprehensive drunken driving laws revealed that sharp initial declines in fatal crashes following enactment of the laws were compromised by a shift in police enforcement, after the enactment, away from speeding enforcement (*16*). State police arrests for speeding declined from 31,000 the year the laws passed to 10,000 2 years later. During that time, the proportion of drivers traveling 65 miles per hour or more on 55 mph posted roads almost doubled. Night fatal crashes, which had declined 33 percent the year after the laws were passed, returned to the previous level by the third year. When Maine officials responded by initiating a speeding crackdown in January 1985, fatal crashes again began to decline, dropping 16 percent in 1985 relative to 1984 (fig. 5).

In a 10-year retrospective study of the impact of the national 55 mph speed limit, the National Research Council concluded that this legislation reduced traffic fatalities initially by 3,000 to 5,000 per year and by 2,000 to 3,000 per year a decade after enactment (*20*). Because of their poor sensory-motor coordination and reaction time, drunken drivers may be particularly vulnerable to crashes at high speed.

Maine's experience unfortunately may be a precursor of the national experience. In 1987, 38 States raised their speed limits to 65 mph on rural interstate highways. A recent U.S. Department of Transportation report indicates that during the first 9 months, traffic fatalities *increased* 23 percent on rural interstates. At the same time, fatalities *declined* 13 percent on urban interstates and 5 percent on other roads in those States where speed limits were not raised (*21*). Benefits of strict drunken driving laws and strict enforcement of those laws may be compromised if States relax their speed limits and their enforcement of speed limits.

Fourth, we recommend the development of programs aimed at increasing informal social pressures not to drive drunk. Too often the public response to traffic problems is to delegate them to the police. An effective but less often pursued deterrent to drunken driving is social disapproval by one's peers. As noted earlier, declines in fatal crashes in many States began prior to the passage of drunken driving laws, as the public debated the problems posed by drunken drivers (*16*).

In addition, other behaviors that contribute to alcohol-related traffic fatalities, such as pedestrian intoxication or riding with a drunken driver, should be targeted. For example, every year approximately 4,000 persons who are passengers riding with a drunken driver are killed in crashes. Getting into a car with a drunken driver indicates tacit acceptance of the driver's behavior. Every effort must be made to persuade people not to ride with intoxicated drivers and to provide alternative transportation.

Social pressure has been shown to mediate risky behavior in other areas. Community-wide, multistrategy interventions for changing behavior related to heart disease risk factors (for example, smoking, diet, alcohol consumption, and sedentary lifestyle) have demonstrated long-term success. Large-scale controlled studies in Finland, California, and Minnesota assessed programs that target high-risk groups and the total community via the media, schools, workplaces, and civic organizations (*22–24*). Significant positive behavior changes were reported across socioeconomic and risk factor strata in the target communities relative to comparison communities. These studies indicate the potential for sustaining behavior change by developing a community consensus about a problem. Establishing this kind of program requires a substantial commitment of time, energy, and financial resources, not only from Federal and State governments, but also from grassroots public and private organizations and local communities. Whether such programs can successfully achieve long-term change

Table 1. Drunk driving enforcement in New England, 1982–85

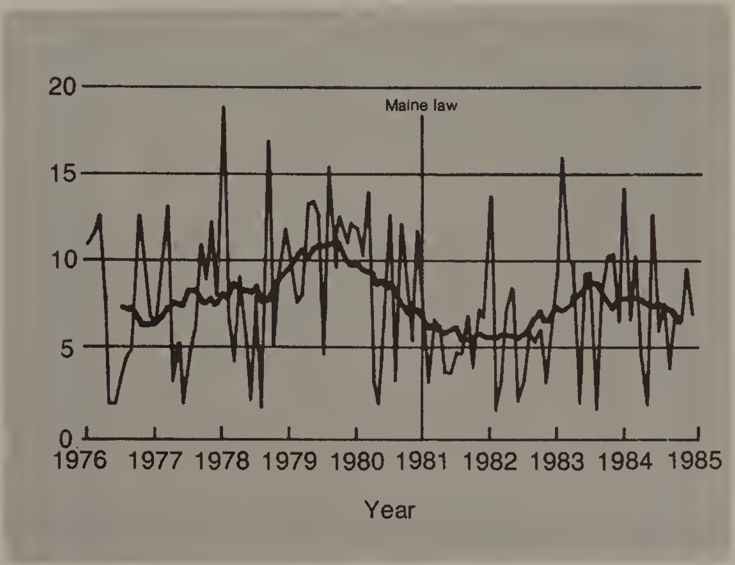
Category	*1982 (N = 3,314)*	*1983 (N = 3,340)*	*1984 (N = 3,248)*	*1985 (N = 3,282)*
Stops after drinking, per 1,000 D.D. trips[1]	11.1	16.1	21.1	33.1
Took Breath test, per 1,000 D.D. trips	2.5	4.6	3.4	5.9
Tickets for DWI, per 1,000 D.D. trips	1.0	2.0	2.0	2.8
Accidents after drinking, per 1,000 D.D. trips	2.1	2.5	3.0	2.1

[1] D.D. trip = trip after 5 or more drinks.

Table 2. Characteristics of persons who drive after heavy drinking (5 or more drinks) according to a survey of adult drivers in Massachusetts, 1985 and 1986, in percentages

	Times drove in previous month after heavy drinking		
Other risky behaviors	*0 (N = 1,758)*	*1–3 (N = 88)*	*More than 4 (N = 30)*
Previous month			
Drove after using marijuana	4	18	41
Drove after using other drugs	3	4	27
Wore seatbelts 90 percent of the time	43	22	11
Previous week			
Ran a red light	8	12	18
Speed 20 mph over limit	15	39	32
Previous year			
Driving violation other than drunk	11	29	40
Driver in crash	9	19	19
Driver in crash resulting in injury	1	2	5
Strongly oppose—			
Seatbelt law	18	26	26
Safe roads act (drunk driving law)	9	17	70
55 mph speed limit	11	12	35

Figure 5. Fatal single vehicle night (SVN) crashes per nighttime vehicle miles traveled and annual fatal and SVN crash frequencies, Maine, 1976-85



NOTE: Smoothed curve is a moving average. Years shown are September through August.

in social norms about drunken driving warrants careful study.

Finally, in addition to measures that aim to separate drinking and driving, policies can restrict the availability of alcohol to reduce drinking, restrict access to vehicles among groups likely to drink, and make vehicles and roadways safer and thereby reduce alcohol-related traffic deaths (see box).

Taxation, dram shop laws, minimum drinking ages, server and host liability, land use laws governing the location of alcohol outlets, and prohibitions on advertising alcoholic beverages can affect the driver's access to alcohol. Increasing taxes on alcohol has been associated with reduced drinking, specifically among teenagers (*25–27*), and with declines in fatal crashes (*28*).

Also, States that delay licensure among teenagers have fewer traffic deaths among the targeted age groups, without any apparent increase in the rate of fatalities once teenagers reach driving age (*29,30*). Teenagers are overrepresented among nighttime traffic fatalities, and night fatal crashes disproportionately involve alcohol. In recent years, 12 States have adopted nighttime driving curfews for young drivers, and curfews have been shown to result in substantial reductions in crashes among 16-year-olds (*31*). Expanding public transportation or otherwise providing alternatives to automobiles, in conjunction with measures that restrict access to vehicles among young, high-risk drivers, might further increase the effectiveness of driver age and curfew restrictions.

In 1984, more than 90 percent of vehicle occupants killed in crashes involving drunken drivers were not wearing seatbelts. Since that time, 33 States, representing over 80 percent of the U.S. populations, have adopted mandatory seatbelt legislation. It has been estimated that by the end of 1986, occupant deaths declined 5 to 10 percent in States that had seatbelt laws compared with States that did not have such laws, saving 1,000 lives (*32,33*). However, because there is evidence that persons who drive drunk are also least likely to adopt seatbelt use (*33,34*), the potential benefits of these laws on reducing drunken driving fatalities may not have been fully realized.

In the 1960s, Federal laws established vehicle design safety standards. Included were requirements for the installation of seatbelts, energy absorbing steering columns, head restraints, and side door beams. Several studies suggest that these interventions have been effective. An analysis by Graham and Garber suggests Federal safety standards for vehicle construction have resulted in reductions of 15 to 35 percent in traffic fatalities (*35*). A study by Robertson concludes that some 37,000 fewer deaths occurred between 1975 and 1978 than would have been expected without the Federal standards (*36*). The effectiveness of roadway design in preventing traffic fatalities is suggested by the fact that the crash rates on interstate highways are considerably lower than on secondary roads (*2*). The full benefits of new safety standards, such as those requiring airbags or other automatic restraints in new vehicles, may take several years to realize, however, because those persons most likely to drive after heavy drinking tend to drive older cars (*37*).

Conclusion

Some researchers have hypothesized that low levels of police enforcement and difficulties inherent in the ability of police to identify drunken drivers compromise the long-term potential for increased penalties or measures to facilitate court conviction to deter drunken driving (*1*).

However, we contend the drunken driving laws of the 1980s did not occur outside of a larger social context, nor do they exert their effects only through fear of apprehension and punishment. Laws are an expression of emerging social norms. In this sense, they are statements by society of new standards of acceptable behavior (*38*). In responding to changes in social norms, as opposed to only fear of apprehension, the individual can become the monitor of his or her own behavior and, through individual actions and attitudes, the disseminator of behavioral change. Peers can also monitor each other's behavior and exert influence by confirming new norms.

We suggest that drinking and driving laws should be viewed within the context of the development of social norms about the appropriateness of drinking and driving. The process begins with the perception of drunken driving as a social problem and progresses to social activism and the formation of the political consensus required to enact legislation. Public debate about the law raises the issue in the public's consciousness and may independently stimulate behavior change. The legislation itself can have the immediate effect of deterring drunken driving by reason of fear of apprehension. This deterrent effect, however, can dissipate in the long run, particularly if media attention to the law and enforcement of the law slacken. But the effects of the law in anchoring new social attitudes and norms may continue.

This sequence of events is not, of course, predestined. Public concern about drunken driving may wane before social norms become firmly established. We have seen, for example, a gradual erosion in compliance with the 55 mph speed limits, and in 1987, 38 States passed legislation raising speed limits on some of their roads. New norms about drinking and driving are not inevitable, but the process of perceiving a social problem and of enacting and enforcing legislation to address the problem must be viewed within the context of changing public attitudes and beliefs.

Viewing the law as an element in the process of social change has implications for how we implement and evaluate drunken driving legislation. To the extent that laws function to disseminate and reinforce social norms about drunken driving, outcome measures of the impact of laws should not only examine fatal crash trends, but they should also examine attitudes about the laws and their enforcement, perceived dangers of drunken driving, and social pressure not to drive drunk, as well as behavioral variables. The periods of pre- and post-law observations should be extended to allow time for normative change to occur and to observe when it occurred relative to the passage of the law. This extension would permit an assessment of whether the law initiates or reinforces normative change.

If laws contribute to social change by way of the evolution of behavioral norms, then the implementation of these laws should be accompanied by activities aimed at accelerating social change. Legal interventions should be accompanied by increased police enforcement as well as educational programs and community organization aimed at all strata of the driving population. Those programs should heighten informal social pressure not to drive drunk and should disseminate information about behavioral alternatives to drinking and driving.

Strategies to reduce traffic fatalities and injuries associated with drunk driving

Make alcohol less accessible
Raise drinking age
Higher taxes
Zoning restrictions on sales
Happy hour laws

Make vehicles less accessible or necessary
Raise taxes on vehicles
Increase public transportation
Raise driving age
Curfews
Interlock system for driver

Make driving environment safer
Make crashworthy vehicles
  airbags
  automatic seat belts
Reduce speeds
Widen lanes, better marking, banking
Fewer two-lane, high-speed roads
Seatbelt laws
Improve EMS and medical care

Dissuade drivers from drinking
Education
General deterrence
Increased fines
Greater police enforcement
Longer license suspension
Increase convictions

Specific deterrence
Treatment and re-education
License revocation, etc.

The legislative reforms of the 1980s, such as raising the legal drinking age, enactment of criminal and administrative per se laws, and increases in penalties, as well as public discussion about drunken driving and its dangers, have all helped reduce drunken driving across the Nation. Progress has been made and further progress can be made in reducing alcohol-related traffic deaths and injuries. But we should not be lulled into complacency simply because efforts in the early 1980s proved successful in reducing the number of fatal crashes. The drunken driving problem will quickly re-emerge if we do not continuously engage in efforts to keep public attention focused on this issue and develop a strong social consensus that driving after heavy drinking and other risks, such as speeding and not wearing seatbelts, are unacceptable behaviors that pose a threat to drunken drivers and to others as well.

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University Students' Drinking Patterns and Problems: Examining the Effects of Raising the Purchase Age

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Synopsis

An extensive review of the literature on college students' drinking patterns and problems since the mid-1930s revealed no radical changes over the past several decades. However, during the past 10 years, drinking and problems related to drinking and driving have gradually decreased among college students.

Results of a study of students at the same 56 colleges and universities throughout the United States (3,145 in 1982–83, 2,797 in 1984–85, and 3,375 in 1987–88) revealed few changes in collegiate drinking patterns and problems attributable to the nationwide increase in the minimum age for alcohol purchase. There was a decline in the proportion of students who drank in the period during which the law changed. However, the proportion of students categorized as heavy drinkers remained constant over time and the proportion of underage students (81 percent) who drank was higher than the proportion of legal age students who drank (75 percent). Of 17 problems related to drinking, all but 5 remained stable over the time periods. Three of the problems represent the continuum of an established trend of fewer students to indicating drinking and driving-related problems.

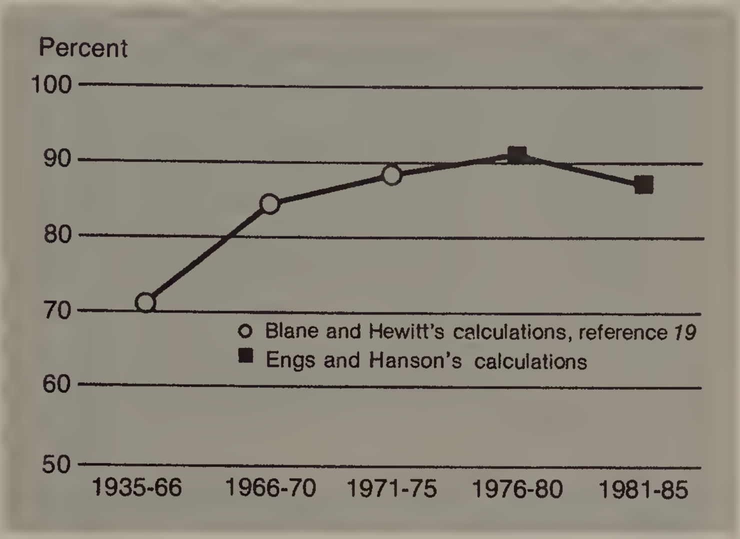
As discussed in this paper, creative alcohol programming can assist in controlling alcohol abuse among college students.

COLLEGE STUDENT DRINKING, legal or illegal, is not a recent occurrence in the United States. As far back as the early 18th century, alcohol was used by students, and there were admonitions and strict regulations on the part of authority figures regarding the practice. However, students have drunk over time regardless of restrictions or prohibitions (*1*). In spite of laws and their many changes, only gradual changes in collegiate drinking patterns have occurred over the past several decades.

Cross-Sectional Studies

A number of cross-sectional or point-prevalence studies have been conducted over the years within individual universities, States, or regions as well as

Mean percentage of students who drink as reported in the literature, 1935–85



> *. . . 'the per capita alcohol consumption has stabilized or decreased in many western countries since 1975–76, although the proportion of young drinkers has increased through the mid-1970s. On the whole, since the mid-1930s, almost 80 percent of the college population has consumed alcohol and continues to do so.'*

nationwide to determine levels of drinking among college students. Despite the expected differences in sampling procedures, statistical analyses, and definitions of drinking among these studies, certain trends in drinking patterns among college students can be identified.

Early studies from the 1930s through the 1950s, including Straus and Bacon's nationwide survey, reported that the proportion of college students who were drinkers ranged from 56 to 95 percent (*2–6*). During the 1960s and early 1970s, several studies described regional variation in collegiate drinking (*7–13*). Among the findings were these:

- 68 percent of women and 62 percent of men from a western university drank.
- 58 percent of freshmen and 88 percent of seniors at a midwestern university had used alcohol.
- 68 percent of students from a southern university were drinkers.
- 91 percent of students from a large eastern State university system used alcohol.

Several national studies of college students' drinking patterns were conducted in the late 1960s and early 1970s. These studies reported that approximately 80 percent of college students drank (*14,15*). In one of the studies (*15*) it was also found that more than 50 percent of students reported either having a hangover or driving after drinking during the previous 12 months.

Further research in the late 1970s reported on prevalence of heavy drinking and problems related to drinking among college students. Among the New England college students, 29 percent of the men and 11 percent of the women were classified as heavy drinkers (*16*), and among these students, more than half reported problems related to drinking, such as blackouts, fighting, and trouble with authorities (*17*). Strange and Schmidt (*18*) found that 92 percent of the students at the University of Iowa drank and that self-identified problem drinkers were more likely to experience problems related to their drinking than were nonproblem drinkers. (See Blane and Hewitt [*19*] for a comprehensive review of the literature of college student drinking patterns through the 1970s.)

Interest in college drinking has continued into the 1980s. At least 11 surveys (20–30) at individual universities and States have been conducted through 1984. From these surveys, estimates of the proportion of drinkers among college students ranged from 80 percent for the University of South Florida (*22*) to 99 percent for a private college in western New York (*21*).

On the national level, two surveys (*31,32*) conducted in 1982–83 and 1984–85 found little change over the study period in three measures of student drinking: proportion of drinkers, of heavy drinkers, and having hangovers or driving after drinking. Only the proportion of students reporting vomiting, drinking while driving, or driving after they knew they had drunk too much had changed, from approximately 25 percent in 1982–83 to more than 40 percent in 1984–85.

On the international level, various studies (*33–35*) report similar proportions of drinkers among students in Australia (85 percent), Oxford University (82 percent), and Scotland (87 percent).

Longitudinal Studies

From the early 1970s through the mid-1980s, several longitudinal studies (*31,36–41*) have examined changes in student drinking over time with conflicting results. Two studies (*36,37*) reported steady increases through the early to mid-1970s in drinking at the universities studied, while two other studies (*39,40*), one from the early 1970s and the other from the early to mid-1980s, found decreases in drinking among the students surveyed. In contrast, three other studies (*31,38,41*) reported no apparent change over time in the proportion of drinkers on the college campuses surveyed.

Most of these longitudinal studies used different sampling procedures, statistical analyses, and definitions of drinking. Within these limitations, the incidence of drinking during different periods has been calculated based on the mean percentage of students who drink as reported in the literature. The proportion of drinkers apparently changed between 1950 and the present. Blane and Hewitt (*19*) found that the mean percentage of those who had ever drunk alcohol rose from 71.4 percent (1936–65) to 84.3 percent (1966–70) to 88.5 percent (1971–75). Similarly, from the various American studies, the authors have examined the mean percentage of students' drinking and have found an increase to 90.0 percent from 1976 to 1980 and a decrease to 88.2 percent from 1981 to 1985 (see figure).

Since the late 1970s, there has been a general downward trend in overall alcohol consumption. According to Smart and Murray (*42*), the per capita alcohol consumption has stabilized or decreased in many western countries since 1975–76, although the proportion of young drinkers had increased through the mid-1970s. On the whole, since the mid-1930s, almost 80 percent of the college population has consumed alcohol and continues to do so.

Drinking in Relationship to Age

Many studies have examined drinking patterns and problems in relation to age. Studies from the 1950s and 1960s (*3,8,14,43*) suggest that older students drank significantly more than younger students, and the proportion of students who drank appeared to increase as they progressed through college. Beginning in the mid-1970s through the mid-1980s, many studies (*15,16,26,44*) reported few age-related differences in the proportion of drinkers, heavy drinkers, or problems associated with drinking.

Several recent individual university, regional, and national studies (*25,30–32,45–48*), however, have reported that younger students are more likely to exhibit problems related to drinking, including heavy drinking. Although older students are more likely to have had a drink (by as much as 15 percentage points [*30*]), they are also more likely to drink in moderation as they progress through college.

Collectively, these reports appear to suggest that increasingly since the mid-1970s, students have begun college with drinking patterns and problems already established and, as they mature in college, are less likely to exhibit heavy drinking and drinking problems. As students progress through college, they are more likely to drink, but to do so in moderation, and are less likely to experience drinking problems.

Legislation and Drinking

Over the past two decades, many changes have been made in the drinking laws of various States that have affected university students and the general youth population.

All States in the United States were required to establish 21 years as their minimum alcohol purchase age no later than July 1987 if they wished to continue receiving Federal highway funds. Since the early 1970s, the legal drinking age in a number of States has changed. Some studies (*44,49*) have found little difference between drinking patterns of students who were legally able and not legally able to drink. One reason for this lack of differentiation may be that, even if a State had raised its legal drinking age, students were often able to drive to neighboring States with different drinking laws to purchase or consume alcohol.

In 1970, Congress passed the Twenty-Sixth Amendment to the United States Constitution, which granted the right to vote in Federal elections to citizens between the ages of 18 and 21. A movement then began to extend other rights and privileges of adulthood to those aged 18; between 1970 and 1975, 29 States reduced their minimum legal drinking age (*50*).

Traffic crashes have been examined in studies of the effects of changing the minimum drinking age. A significant increase in auto accidents among those affected by lowered minimum drinking age laws has been reported in virtually all studies. Other research has examined the effects of such legislation on drinking patterns and problems of young people. Again, virtually all studies report increased consumption or greater drinking problems, or both, in States with a lower drinking age. For excellent reviews of the research literature, see

'The proportion of students who drank declined in the period when there were major nationwide increases in minimum purchase age laws. On the other hand, the proportion of heavy drinkers remained constant over time, and the proportion of underage students who drank was actually higher than the proportion of legal-age students who drank.'

Wagenaar (*50*) and a General Accounting Office report (*51*).

Hypotheses

Based on the literature, which suggests that the proportion of students who consumed alcohol gradually increased during the 1960s and 1970s and slightly declined during the 1980s (before nationwide legislation was posited), the following null hypotheses were suggested. Before and after a national increase in the minimum age permitting the purchase of alcohol in the United States, no significant difference in drinking patterns or in drinking problems would occur among college students.

Method

Instrument. A precoded instrument, "The Student Alcohol Questionnaire" (*52,53*), was used. Also used by hundreds of other researchers over the past 15 years, it has an internal consistency reliability of .79 and includes demographic items, questions regarding the consumption of various alcoholic beverages, and 17 items concerning possible consequences of drinking. All question items had been used by the authors in the two previous periods with which the current sample is compared. The instructions for the instrument explained the voluntary nature of participation in the study as approved by the Indiana University Human Subjects Committee.

Sample. All colleges and universities that participated in the 1982–83 and 1984–85 studies were contacted. The resulting sample contained 56 universities that could be matched over the three periods. These represent 4-year colleges and universities in all States. If the same university was not available for a given period, a similar college matched for size and funding source from the same State was used.

The sample was drawn, as in the earlier studies by the authors, from administration of the questionnaire in survey-type sociology and health or physical education courses that had a high probability of containing students from every academic major and class year during the 1987–88 academic year. The response rate exceeded 98 percent, and the demographic composition of the resulting sample of 3,375 closely approximates that of the earlier studies as well as the universe of students attending baccalaureate institutions of higher learning in the United States. The sample for the 1984–85 period was 2,797 and for the 1982–83 period was 3,145. Because of its very large size, the sample had high power for detecting significant differences. The .001 level of significance was selected to rule out type 1 errors due to the large sample size.

Results

Drinking patterns. A significant ($X^2 = 52.56$, $df = 10$, $P < .0001$) decrease occurred in the proportion of students who were drinkers in 1985–86 (83 percent) compared with 1987–88 (79 percent). A drinker was considered someone who had consumed alcohol at least once during the previous 12 months. The decrease occurred in the period when there were major nationwide changes in the laws, increasing the minimum age for purchases of alcohol. No change occurred in the proportion of drinkers between 1983 and 1985.

The decrease in drinkers between the 1984–85 and the 1987–88 periods occurred because there were fewer light to moderate drinkers. Heavy drinkers (those who consume six or more drinks at any one sitting more than once a week) remained a constant 20 percent of the total sample. Among drinkers, 25 percent from all three periods were heavy drinkers (table 1).

Moreover, analysis of the 1987–88 sample revealed that a significantly ($X^2 = 16.8$, $df = 1$) higher proportion of underage students drank (81.2 percent) compared with legal-age students (75.3 percent).

Drinking problems. Of 17 identified drinking problems, significant ($P < .001$) decreases were noted in 3 of them over the three periods: the proportion of students indicating that they "drank while driving a car" ($X^2 = 59.7$, $df = 2$), had "driven a car after drinking" ($X^2 = 23.6$, $df = 2$), and had "driven after they knew they had had too much to drink" ($X^2 = 54.8$, $df = 2$). A significant ($P < .001$) decrease in vomiting as a result of drinking occurred during the 1987–88 period ($X^2 = 17.1$, $df = 2$). However, a higher proportion of students had "gotten into a fight after drinking" ($X^2 = 16.4$, $df = 2$) (table 2).

Discussion

The first hypothesis (no significant differences in collegiate drinking patterns would occur over time) received only partial support. The proportion of students who drank declined in the period when there were major nationwide increases in minimum purchase-age laws. On the other hand, the proportion of heavy drinkers remained constant over time, and the proportion of underage students who drank was actually *higher* than the proportion of legal-age students who drank.

The second hypothesis (no significant differences in collegiate drinking problems would occur over time) received only partial support since only 5 of the 17 drinking problems significantly changed. Three of these appear to represent a continuation of earlier established trends. However, the only drinking problem that appeared to decrease *after* the increased minimum age laws (and hence might not represent, at least in part, a continuation of earlier established trends) was that of "vomiting" as a consequence of drinking. The increase in "fights" during this time perhaps is a statistical anomaly since it could not be adequately explained.

Although the percentage of students who drink has decreased, the proportion of students categorized as heavy drinkers has remained constant. Many reports have indicated that heavy drinkers are those who are most likely to have drinking-related problems. This stability in heavy drinking may explain why few changes occurred in problems related to drinking over the three periods. Even though fewer students drank, the ones who did drink were just as likely to be heavy drinkers as before the change in the laws and to have alcohol-related problems. Persons who are light drinkers, particularly if underage, may be more inclined to obey the law.

A decrease in the proportion of students who drink appears to support other studies completed over the 1980s that show a decline in drinking. This decrease may not be attributed to the national

Table 1. Percentage of students who drank, according to drinking pattern

Drinking pattern	*1983 (N = 3,145)*	*1985 (N = 2,797)*	*1987 (N = 3,375)*
Abstainer [1]	16.2	17.3	[2] 21.2
Infrequent drinker [3]	8.2	8.3	10.2
Light drinker [4]	12.0	11.7	10.2
Moderate drinker [5]	18.7	18.6	17.9
Moderate/heavy drinker [6]	24.0	23.3	20.1
Heavy drinker [7]	20.9	20.8	20.4

[1] Drinks less than once a year or not at all.

[2] $P < .001$.

[3] Drinks more than once a year but less than once a month.

[4] Drinks at least once a month but not more than 1 to 3 drinks at any 1 sitting.

[5] Drinks at least once a month with no more than 3 to 4 drinks, at least once a week with no more than 3 to 4 drinks, or at least once a week with no more than 1 to 2 drinks, at any 1 sitting.

[6] Drinks 3 to 4 drinks at least once a week or 5 or more drinks at least once a month.

[7] Drinks 6 or more drinks at any 1 sitting more than once a week.

Table 2. Percentage of drinkers who experienced drinking problems during the preceding year

Drinking problem	*1983 (N = 2,637)*	*1985 (N = 2,313)*	*1987 (N = 2,657)*
Had a hangover	72.5	73.8	75.7
Vomited as a result of drinking	44.2	46.4	37.1 [1]
Driven a car after having had several drinks	40.4	38.0	34.0 [1]
Driven a car when they knew they had drunk too much	59.8	55.3	49.2 [1]
Driven a car while drinking	47.4	42.5	37.3 [1]
Came to class after having several drinks	9.0	8.6	8.4
"Cut a class" after having several drinks	9.3	10.8	9.1
Missed a class because of a hangover	23.5	27.6	26.5
Been arrested for driving while intoxicated	1.6	1.1	1.6
Been criticized by someone they were dating because of their drinking	11.5	10.7	13.4
Had trouble with the law because of drinking	4.9	3.7	6.2
Lost a job because of drinking	0.3	0.3	1.0
Gotten a lower grade because of drinking too much	4.4	5.8	5.4
Gotten into trouble with school administration because of behavior resulting from drinking too much	2.0	1.8	2.9
Gotten into a fight after drinking	11.1	12.4	14.7 [1]
Thought they might have a problem with their drinking	9.2	8.1	9.9
Damaged property, pulled a false fire alarm, or other such behavior after drinking	9.4	9.4	11.3

[1] $P < .001$.

change in the laws regulating purchase of alcoholic beverages but may represent a continued national and international trend that has been observed by others (*42*). Likewise, the three drinking and driving variables appear to be the continuation of a trend that began before the law was changed.

Implications

Drinking patterns of college students have been of increasing interest to college administrators as they attempt to reduce alcohol-related problems on campus (*54*). Unfortunately, the results of most alcohol education and programming efforts have been disappointing (*13,55-59*).

Based on the results of this study, however, campus administrators are recommended to develop comprehensive and practical alcohol education programming for college students. Although it is illegal for much of the student population to drink, clearly about 80 percent of students, including those who are underage, drink at least once a year. At least half of all students have experienced negative problems related to drinking.

Creative programming that emphasizes the concept of interesting nondrinking activities and events, safe planning and hosting of parties, responsible choices concerning drinking, assertion skills, self-responsibility, and the mature use of alcohol for those who do choose to drink need to be included in any educational efforts aimed at the college-age population (*55,56,60,61*). Simply telling college students that it is illegal to drink and that they will be punished if they break the law is unlikely to have a significant impact on drinking or alcohol abuse problems.

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An Overview of Prevention Research: Issues, Answers, and New Agendas

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Synopsis

Efforts to curtail alcohol abuse and alcoholism can be divided into primary, secondary, and tertiary prevention. Primary prevention attempts to stop a problem or illness from occurring in the first place. Secondary prevention identifies persons in the early stages of problematic or illness behavior and refers them for counseling or treatment, which is considered tertiary prevention. Five research areas concerned with primary and secondary prevention are selected for discussion: youth, the mass media, the worksite, blacks and Hispanics, and alcohol-related behavior that increases the risk of AIDS. Several of these themes have been in the forefront of alcohol prevention research; others such as AIDS are emergent areas of inquiry.

The discussion to follow briefly summarizes research approaches, key findings, methodological shortcomings, and suggested issues for future investigation. Although scientifically solid prevention studies have been conducted, more rigorous, more comprehensive, and more innovative research is needed. Given the dynamic sociocultural and economic systems in which prevention occurs, research techniques that can address this complexity are required. A range of appropriate methodologies is described.

BECAUSE OF social and moral imperatives, alcohol abuse and alcoholism in the United States are continuous catalysts for counteractions aimed at reducing and minimizing the deleterious effects of alcohol problems. Systematic and episodic prevention activities are inherently part of the larger sociocultural dynamic that promotes alcohol use and abuse, on the one hand, and alcohol control, on the other. Responsibility for the prevention of alcohol abuse rests officially with governmental agencies, the alcohol industry, and consumers of alcohol; but populist movements and organized constituencies within the citizenry at large also play critical roles in designing and implementing prevention strategies.

Ideally, interventions to reduce the incidence and prevalence of alcohol-induced problems should be based on scientific evidence of the effectiveness of the proposed interventions. Proof of benefit, net benefit, absence of benefit, or actual harm can have pragmatic as well as theoretical implications for the prevention process. This information can contribute to and influence the dialogue and debate on appropriate strategies for action, adding objectivity and rigor to the decision-making endeavor. However, the armamentarium of evidence favoring or disfavoring particular interventions is still in the stage of early development. Much remains to be learned about the short- and long-term effectiveness of various social technologies for inducing behavioral change. The extent of the knowledge base depends on the type of prevention approach and the nature of the target group.

In the absence of proven prevention strategies, community pressure to curtail alcohol abuse may necessitate the selection of interventions on the basis of prudence rather than proof (*1*). Since prudence is the exercise of sound judgment, those who shape prudent prevention policies must be guided by the weight of available evidence. It is incumbent on them to consider research findings concerning the benefits, risks, and costs of tested approaches to prevention. Strategies that signal promise of eventual success deserve special attention, and failures can be teaching experiences in their own right.

Alcohol prevention research focuses on a wide range of endpoints related to drinking behavior, addiction, sequelae of alcoholism (such as cirrhosis), and deleterious consequences of alcohol abuse (such as traffic accidents, violence, and absenteeism). The outcome measure depends to a large degree on the target problem, target population, and choice of intervention. For example, mortality can serve as an endpoint for strategies to prevent alcohol-induced traffic accidents (*2a*) but not for interventions aimed at reducing fetal alcohol syndrome (*2b*).

Efforts to prevent alcohol problems can be divided into primary, secondary, and tertiary prevention (*3*). This article is concerned only with primary and secondary prevention. Primary prevention attempts to stop the problem or illness from occurring in the first place; secondary prevention identifies persons in the early stages of problematic or illness behavior and refers them for counseling or treatment. (Tertiary prevention consists of the actual counseling or treatment.)

The discussion to follow summarizes prevention research in five selected areas: youth, the mass media, the worksite, blacks and Hispanics, and alcohol-related behavior that increases the risk of AIDS. Each of these areas offers rich opportunities to enhance the state of science in prevention research. Topics addressed elsewhere in this issue are deliberately not considered here, including research on traffic safety and State and local laws that control alcohol availability and the minimum drinking age.

Youth and Young Adults

Epidemiologic analyses reveal that alcohol use and abuse constitute a serious problem among youth and young adults. (*2c,2d,4*). This problem is expressed in the preadolescent onset of drinking behavior, heavy drinking among high school and college students, and the overrepresentation of legal intoxication among young males involved in fatal accidents (*2d,2e,4*).

A growing public awareness and social concern have led to the development of a variety of prevention strategies, many designed for school settings (*2c,2f,5*). Some strategies are directly or indirectly theory-based, while others spring from reasoned hunches or intuition (*6,7*). Three intervention models are used: increasing knowledge and changing attitudes, teaching values and decision-making skills, and developing social competency skills (*2f*). Studies of the effects of prevention frequently blur distinctions between alcohol, cigarettes, and other drugs, making it difficult to measure the impact of the interventions on alcohol use per se (*5,8*).

Evaluations of alcohol-targeted prevention strategies, including two recent meta-analyses, suggest that school-based interventions have not been particularly effective, especially the more traditional didactic approaches (*2f,5–11*). In some cases, an increase in students' knowledge about alcohol has even led to increased use of alcohol (*5–10*).

The newer approaches (teaching peer refusal and social skills, correcting perceptions of social norms) show some promise but lack uniform success (*2f,6–10*). Attitude and behavior change is more substantial in the highest intensity, multimodal programs, using peer or parent leaders and "booster" sessions in later years (*2f,6–10*). Although costly, so-called "alternatives programs" (that provide healthy opportunities for stimulation, recognition, and learning) show moderate success for special population groups, such as drug abusers or juvenile delinquents (*9*). Prevention efforts on college campuses have yielded variable yet encouraging results (*2f,12–17*). Multi-session efforts, preferably coupled with field experiences, have resulted in actual changes in drinking patterns and in drinking and driving behavior (*12–14,17*).

In assessing and interpreting reported studies, one confronts a number of methodological problems. The absence of a consensus regarding prevention goals (for example, abstinence versus moderation) can confuse or constrain the choice of outcome measures (*5,7,11*). Illustrations of other methodological difficulties include the lack of appropriate comparison groups (*5,6,8,9,11*); inadequate or biased samples (*5–9,11*); lack of pretest, posttest, or followup measures (*5,6,8,11*); high attrition rates (*5–7,11*); questionable validity of the dependent variable (*5–8,11*); questionable validity of the dependent variable (*5–8,11*); absence of outcome linkages to behavior (*5,6,8,9,11*); and inappropriate statistical analyses (*5–9,11*). Some studies have also been criticized for selective reporting and self-serving interpretations (*8,11*).

Recommendations for enhancing research utility include longer followup periods to determine impact durability and delay (*5,7,8*); measurement instruments that provide better reliability, validity, and comparability (*5,7,11*); examination of effects on various subpopulations (*5,7–9,11*); thorough process evaluation and implementation assessment (*7,8,11*); additional replication studies; and, possibly, more secondary analyses (*7,8,11*) and archiving of data (*8* and Moskowitz, J., Berkeley, CA, personal communication 1988). Suggested foci for prevention strategies include (*a*) adapting interventions to particular developmental stages (*5,7,9*); (*b*) addressing adolescent risk-taking behaviors (*5,7,18*); (*c*) utilizing parents as teachers of normative behavior (*7,8*); and (*d*) developing special strategies for nonschool youth (*5,7*) and the community (*7,10,11*).

Advertising and Media

The literature on media and alcohol consumption reveals two central research questions: Does advertising by the alcohol industry influence the use or abuse of alcoholic beverages? And do prevention-oriented media campaigns and countermeasures influence drinking behavior?

Controversy surrounds the extent to which alcohol advertising and marketing affect the American public. Some suggest that advertising increases overall consumption (*19,20*). Others contend that it simply causes the already drinking public to shift from one brand or type of alcoholic beverage to another (*21–23*). Results of relevant research are inconclusive.

A major issue that needs to be addressed in future research is how to separate the overall impact of alcohol advertising from the broader social context of drinking (*24*). The Board of Trustees of the American Medical Association (AMA) recommended that "additional well-designed research be conducted under impartial and independent auspices to provide more definitive evidence on whether, and in what manner, advertising contributes to alcohol abuse" (*25*). Research results could have implications for public policy concerning alcohol advertising. The AMA has noted that no Federal legislation on this issue has been enacted since the repeal of Prohibition in 1933.

In addition to the billion dollar a year advertising campaigns of the alcohol industry, television programs themselves may inadvertently promote a positive perception of alcohol consumption (*26*). Content analyses of television programs indicate that alcohol consumption is depicted as acceptable and glamorous and that realistic and undesirable potential consequences of alcohol abuse are rarely shown (*26, 27*).

It appears that the media are most effective in imparting knowledge, less so in changing attitudes, and least effective in changing behavior (*28–30*). Media campaigns seem to be particularly effective when combined with some type of interpersonal communication (*3*), as occurred in Project CRASH (with informal conversation) and the Stanford Heart Study (with structured group training) (*28,31*). Of course, labor-intensive programs can increase costs.

Although television is obviously a popular medium (*26*), successful prevention campaigns may need to involve several types of media. Both the message and the medium should be specifically tailored to the target population (*30*). As examples, radio may be better than television in reaching college students (*28,29*), and billboards may be especially useful in reaching the driving population.

The full potential of the media as a prevention strategy has yet to be determined. The same kinds of problems that typify studies of youth and alcohol (for example, the lack of a theoretical base and inadequate samples, designs, and methods) have also characterized research on the media. There is a particular need for sophisticated designs that will permit causal inferences to be drawn (*28*).

Worksite Research

The worksite can only be fully understood within the context of the larger society and economy. Social scientists have long been aware of the potential effects of economic conditions on the mental health of the work force. For example, Brenner (*32*) demonstrated that an inverse relationship exists between the state of the economy and various kinds of mental health problems, including alcoholism. Moreover, the choice of prevention strategies in the workplace reflects the social climate and ideologies of contemporary society. One current illustration is the expansion of prohibitions against smoking on the job.

Historically, the worksite has been permeated by various movements and innovations designed to improve employee productivity and efficiency. Some of these have focused on changing the individual; others have focused on changing the work environment. One such innovation is the Employee Assistance Program (EAP). The twofold goal of the EAP is to improve employee performance while simultaneously attending to alcohol, drug abuse, and mental health problems among workers.

Researchers and policy makers have searched for causes of alcohol problems in the workplace. Some contend that the workplace may contribute to the development of drinking problems (*33*) while others (*34–36*) claim that employees bring these disorders with them into the workplace. Each perspective has received attention in the models that have been suggested to guide research on workplace alcohol problems (*37–41*). Among these models are those relating to workplace culture, social control, alienation, social stress, and availability. The rapid growth, expansion, and investigation of EAPs over the past decade have resulted in an emphasis on the social control model. Proponents of this model (*35,42,43*) claim that "research supports the idea that progressive, positive discipline, containing expressions of emotional support and offers of help, combined with realistic confrontations about poor job performance, is an effective deterrent to alcohol problems" (*41*).

Primary and secondary prevention of alcohol abuse at the worksite can occur in many ways. It can come about informally "as in the Sandhogs where AA members say, 'you do not have to be drunk to do this job' " (*41*). It also can take place administratively through the adoption and implementation of normative standards concerning a variety of issues: limiting the availability of alcohol on the job and in the adjacent community, refusing to reimburse expenditures for alcohol, having alcohol-free company parties, and discouraging the abuse of alcohol in nonwork settings.

Secondary prevention has been the major focus of alcohol research at the worksite. Although it is important to advance this line of inquiry, it is also critical to stimulate innovative worksite research in primary prevention. One testable question is whether EAPs can play a facilitating role in primary prevention or if a completely different labor-management orientation is required.

Blacks and Hispanics

Blacks compose about 12 percent of the United States population, and Hispanics number more than 6 percent (*2g*). Despite the sizes of these two ethnic minorities, relatively little research has focused on their respective use and abuse of alcohol (*2g,44–48*). Much of the limited literature on prevention strategies among these groups is merely descriptive or impressionistic (*47*). In general, neither the epidemiologic nor the prevention efforts have addressed the wide cultural diversity within the black and Hispanic populations (*46–50*).

A review of medical and psychosocial difficulties reveals that, except for drunken driving, black men reported higher rates of problem drinking than white men (*2h*). Among Hispanic males, about 18 percent reported having one or more alcohol-related problems (*2h,50–52*). Moreover, mortality rates associated with drinking (rates for cirrhosis of the liver, various cancers, and unintentional injuries) are higher in the minority populations than in the general population (*50,53,54*). It is interesting to observe that although alcohol consumption among white men was found to be high for the 18- to 29-year olds and then declined after age 30, consumption among blacks was relatively low for the 18- to 29-year-olds but rose sharply among those in their thirties (*55*).

> *'Prevention strategies should be designed with strong evaluation components to determine what is effective and for whom. Careful attention must paid to the cultural values and social norms of the target population.'*

Prevention strategies are discussed in the literature (44–46,52,56–61) but not from a research perspective. In particular, there is a dearth of information on programs that have been evaluated and found effective (*47,48*). In one study, the reaction of black parents to three popular parent-training programs was tested; many of the components were not accepted by the lower-income parents, who emphasized discipline as the vehicle to respect and obedience (*2i*). Although education efforts are necessary (*62*), they have not been found to be successful by themselves as a prevention strategy (*46*). Thus, in two Hispanic communities in California, an intervention project that relied heavily on a media campaign failed to prevent alcohol problems (*63*).

Recommendations in the literature for new directions in prevention research highlight the need for epidemiologic studies of alcohol use and abuse that recognize the diversity within black and Hispanic populations (*44–48,50*). Prevention strategies should be designed with strong evaluation components to determine what is effective and for whom (*45–48,50*). Careful attention must be paid to the cultural values and social norms of the target population (*2j,44–48,50,64–67*). For example,

> *'Alcohol may have a direct impact on exposure to HIV (and its transfer) through the mechanism of disinhibition—that is, a disregard for social norms, moral obligations, and behavioral restraint.'*

among Hispanics, the influence of the Catholic Church should be taken into consideration (*50*) along with the strong Hispanic commitment to family and community (*44,46,58*). Likewise, religious beliefs and practices and the family play significant roles among blacks (*45–48,68*). Cultural heritage and pride may also represent effective dimensions of prevention efforts (*46,50,68,69*), and socioeconomic and environmental factors cannot be ignored (*44–47,50,55,70,71*). One specific area of inquiry might be the development and testing of prevention strategies to counter the influence of liquor advertising that is aimed at minority groups (*46,70,72*).

Alcohol and AIDS

Studies of patients with acquired immunodeficiency syndrome (AIDS) and cohorts at risk of AIDS suggest that the use and abuse of alcohol may increase the chance of infection from the human immunodeficiency virus (HIV) that causes AIDS (*73–79*). Associations between alcohol and AIDS may in part reflect biological processes whereby alcohol compromises the capacity of the immune system to resist the onset and progression of infection (*80,81*). Possibly more important, however, might be the contribution of alcohol to unsafe sexual practices and drug use.

Alcohol may have a direct impact on exposure to HIV (and its transfer) through the mechanism of disinhibition (*79,81,82*)—that is, a disregard for social norms, moral obligations, and behavioral restraint. Alternatively, the relationship between alcohol and AIDS may be indirect through such processes as selection, attribution (*83,84*), and environmental juxtaposition. Thus, people who abuse alcohol may take additional risks in their sexual or drug-use behavior without any cause and effect relationship, but simply a correlation through selection. Excess drinking also may be deliberately undertaken as an excuse (attribution) for behavior that could not otherwise be explained or rationalized, such as indiscriminate sex. The pretext may actually be self-fulfilling if disinhibition as an anticipated excuse becomes a reality. Finally, it is important to recognize the instrumental function of the tavern or bar as an environment for meeting and entertaining potential sexual partners or making drug connections (*79,85–87*).

The National Institute on Alcohol Abuse and Alcoholism is encouraging research on alcohol-related behavior that increases the risk of AIDS (*88*): studies that explore the nature and dynamics of these behaviors, or that test the effectiveness of prevention strategies, or both. Studies of gay men indicate that users and abusers of alcohol and other drugs during sexual activity constitute a hard-core group that is particularly resistant to adopting and maintaining safe sex techniques (*75,78,79*). Unprotected sexual activity among the heterosexual community (*89,90*) may also be associated with or aggravated by alcohol and drug use (*9*).

One approach to AIDS prevention might be anticipatory education, alerting alcohol users and abusers to their high-risk situation (*75,76*). It may be additionally necessary to confront such issues as the perceived benefits of alcohol for sexual arousal (*75,79*), alcohol use as excusatory behavior (*75*), and the implications of the bar as a context for social interaction. Alcohol-free alternatives have been proposed (*75*). Where there is dependency on alcohol, it may be essential to address the problem of alcohol addiction per se, as well as its relevance to AIDS. In this respect, expanded treatment of intravenous drug abuse has been advocated as a prevention strategy for AIDS (*92*), not simply education regarding sterilization of injection equipment.

Research Methodologies

A variety of research designs and analytical strategies have been applied to prevention research concerning alcohol problems. Qualitative and quantitative methods have both proved fruitful, including ethnographic observations of drinking behavior in natural settings. Examples of ethnographic-observational research are studies of blue collar workers and family drinking (*93*), public drinking and drinking contexts (*94–98*), and worksite behavior (*99*).

Social and health problems associated with alcohol can be viewed in a broad historical perspective. Historical analysis, using both domestic and international data sources, offers promising opportunities for prevention research (*100–103*). Community, school, and worksite prevention trials reflect combinations of several relevant theories and research traditions (for example, learning, organization, communication, behavior change, health education, and medical and social marketing). Interest in such designs has been stimulated by the success of health promotion programs to reduce heart disease (*104,105*). Such approaches have also seen limited use in studies of community interventions for alcohol problems at schools (*106*), studies of local availability of alcohol (*107*), and studies of the mass media (*31*).

Given the difficulty of random assignment in field studies, quasi-experimental designs (*108*) have been used. These designs are often employed in policy analyses of natural experiments, such as changes in alcohol availability. One statistical tool is the interrupted time series analysis (*109,110*), which has more power to deal with problems of autoregression, seasonality, and trending than conventional least-squares regression (*111–114*). Quasi-experimental designs are also employed using nonequivalent control groups or conditions (*108*). These designs frequently rely on multivariate analysis techniques to increase statistical power. Examples include evaluations of server intervention (*115*), happy-hour bans (*116*), prevention among college students (*16*), cross-cultural drinking beliefs, parental influences on adolescent drinking behavior (*117*), and alcohol taxes (*118*).

Studies conducted within the laboratory permit researchers to examine the role that critical experimental factors may play in prevention programs or policies. For example, the potential role of retail price in influencing alcohol consumption has been demonstrated in a laboratory setting in which an actual retail drinking situation is simulated and price is manipulated (*119*). Examples of other analog studies can also be found (*120,121*).

Given the complex and dynamic social, cultural, and economic systems in which prevention occurs, techniques are necessary that can address such complexity. One relevant set of tools is computer modeling. It has particular utility for prevention research because "experiments" can be conducted with the computer to predict potential outcomes prior to expensive field implementation. Examples of applications of computer modeling include Holder and Blose (*122,123*) and Katzper and coworkers (*124*). Econometric modeling has been employed to examine the sensitivity of drinking and alcohol problems to changes in price levels (*125,126*).

One problem confronting prevention research, particularly research most relevant to public policy deliberations, is a need to study real-life situations outside the laboratory. Such research does not provide the precision and opportunity to manipulate variables afforded by laboratory experimentation. Field research is difficult to undertake, often expensive to conduct, and analytically "messy." On the other hand, prevention research undertaken in the laboratory may not be generalizable to the real world because of the controlled, or "hot house," conditions imposed.

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Alcohol Dependency Prevention and Early Intervention

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Synopsis

Current data on efforts to prevent alcoholism indicate that we are better able to prevent some of the consequences of alcohol misuse, such as alcohol-related car crashes and fetal alcohol syndrome, than chronic alcohol dependence itself.

A review of data on outcomes of treatment for long-term alcohol dependence indicates that 9 of 10 alcohol dependent persons receive no treatment for the disorder in any given year. When treatment is provided for long-term alcohol dependent persons, it has only slightly positive results. As a result, many clinicians and researchers have concluded that rather than exclusive preoccupation with long-term alcoholics, early intervention with persons who are just beginning to abuse alcohol may be a more effective use of resources.

ALCOHOL ABUSE and dependence are a major public health problem in the United States. Alcohol abuse is estimated to have cost almost $117 billion in 1983, the latest year for which such data are available. Of this amount, nearly $71 billion was attributed to lost employment and reduced productivity. Another $15 billion was for health care costs. In 1988, these costs will likely approach $150 billion (*1*).

Forty-eight percent of all persons convicted of crimes in the United States in 1983 had been using alcohol when they committed the crime (*1*).

In 1980, alcohol use and abuse was either the main or a contributing cause in almost 100,000 deaths (see table). In 20 percent of these deaths, including those attributable to alcoholic cirrhosis and alcoholic cardiomyopathy, alcohol was the main cause. In an additional 40 percent, those that resulted from alcohol-related automobile and other types of accidents, alcohol was a contributing cause in that death would likely not have occurred had alcohol not impaired judgment and driving ability. In the remaining 40 percent of the deaths, alcohol use accelerated an ongoing physical disease process (*2*).

Alcoholism exacts a terrible, increasing toll in our society that fully justifies our best efforts to prevent and to treat the condition.

Outcomes of Treatment

Data on outcomes of treatment for long-term alcohol dependence are sufficiently discouraging to justify a concentrated search for alternatives. Prevention and early intervention are the most effective alternatives.

In any given year in this country, no more than 10 percent of those who meet accepted criteria for alcohol dependence are treated. These figures include persons whose treatment largely involves contact with self-help groups as well as those treated by professionals. Probably, an even lower percentage of women, minorities, youth, and the

Estimated number of deaths attributable to alcohol, United States, 1980

Cause of death	Number of deaths	Attributable to alcohol: Number	Attributable to alcohol: Percent
Alcohol as main cause [1]	19,587	19,587	100
Alcohol as contributing cause	323,721	77,943	5–50
Cancer[2]	31,955	7,269	20–25
Other diseases	150,280	11,679	5–25
Accidents[3]	96,987	37,849	10–50
Violence[4]	54,499	21,144	30–50

[1] Includes alcohol dependence syndrome, alcoholic cardiomyopathy, alcoholic cirrhosis of the liver, and similarly alcohol-attributable diseases.

[2] Cancer of directly exposed tissues.

[3] Includes motor vehicle traffic accidents, falling, drowning, suffocation, and other alcohol-related accidents.

[4] Includes suicide and homicide.

SOURCE: Reference 2.

elderly, are reached, largely because most treatment programs are designed to meet the needs of the prototypic white, middle-aged, male alcoholic (*3*).

Moreover, as surveys of the treatment outcome literature dating from the early 1970s to the present (*3–5*) continue to show, even when long-term, alcohol dependent persons are treated, treatments are only partly effective.

Despite widely held convictions that the treatment methods used are the ones which work best, the data indicate that different treatment methods do not differ significantly in their effect on the ultimate outcomes of treatment for long-term alcohol dependence. Differences in the theoretical underpinnings of therapies, as well as in the techniques and procedures themselves, do not appear to be associated with differences in treatment outcomes (*5–7*).

The same seems to be true about locus and intensity of treatment. Whether treatment takes place in an inpatient or an outpatient setting, and whether treatment lasts a week, a month, or a year, has not been shown to affect outcomes (*8*). These findings are counter intuitive and unexpected.

Outcomes of treatment for long-term alcohol dependence apparently vary with a number of factors specific to the patient, including age; gender; marital, educational, and occupational status; drinking history and pattern; psychiatric status; and degree of motivation for treatment (*3*, *9*).

In view of the modest results of the national effort to treat long-term alcohol and drug dependency, many of those treating patients have refocused their energies. They are attempting to prevent alcohol and drug problems from developing in the first place, and at the same time are trying to intervene earlier, before emerging problems with alcohol and drugs develop into intractable long-term dependence.

Prevention

Funding. Despite the fact that alcohol and drug abuse are among the Nation's most pressing health and social problems, Federal and State authorities have not recognized the problems by allocating sufficient funds for alcohol and drug prevention. In a recent representative year, for example, only 4 percent of the Federal funds expended on alcohol-related activities were for prevention (*10*). Yet, despite the small size of this prevention commitment in relation to the size of the problem, the funds, $24 million, are 64 percent of the public funds from all sources, Federal, State, and local, expended for alcoholism prevention (*11*).

Both the amount and the percentage of public funds allocated to prevention-related programs for alcohol and drug abusers have increased recently, in response to the belated recognition that the conditions have reached epidemic proportions in this country. However, the percentage of funds for prevention remains substantially less than 10 percent of the public funds earmarked for all alcohol- and drug-related purposes.

One reason that funds for prevention have increased in recent years has been the growing effort to restrict the supply of drugs by trying to destroy them where they are grown and to intercept those who try to bring them into the country. Such prevention methods have been criticized by leaders of the countries where drugs are grown, who fault the United States for failing to enforce its laws against purchase and sale of the drugs, and by American politicians, who claim that the profit margin associated with illegal drugs is so great that it guarantees an adequate supply regardless of any effort to restrict their importation.

Empirical research on the effectiveness of efforts to control alcohol and drug addiction by restricting the availability of the substances suggests that critics of this type of control may be correct. Data indicate that efforts to reduce alcohol and drug dependence by restricting availability are least effective with regard to those persons for whom the efforts are most important, namely those who are heavily addicted (*12*, *13*).

Public education programs. Educational efforts to prevent alcohol and drug abuse in this country are mainly in the form of public education and school-based programs, both of which have achieved moderate results (*14*).

While public education, most often through the mass media, is generally acknowledged to have succeeded in increasing public awareness of the hazards of alcohol and drug misuse, it has probably had little effect on the behavior of those who have developed dependency and are most likely to be disabled by alcohol or drugs.

Public education campaigns have been more successful when they were focused on the consequences of alcohol misuse, such as in drunken driving and fetal alcohol syndrome. In both instances, public pressure generated by public education campaigns has led to changed public attitudes, corrective legislation, increased funding for research and prevention and, most important, decreases in incidence (*15–17*).

School-based programs. Like public education programs, school-based prevention programs are supposed to increase knowledge about and to change attitudes toward alcohol and drugs. In recognition of the presumed malleability of the target population, some programs have been designed to teach values and decision-making skills and to help develop social competence.

Some of these programs have met the goals (*18*, *19*). However, some observers complain that methodological problems prevent an unequivocal interpretation of favorable outcomes (*20*), while others have suggested that attitude clarification and decision-making programs actually undermine the knowledge and attitudes curriculum (*21*). Data that document changes in drinking behavior following full implementation of such programs are rare, as are changes in the incidence of drinking and driving, or changes in the risk of development of alcoholism later in life.

Students who are most responsive to school-based programs probably are those for whom such programs are least necessary. Programs may not be reaching those children who are at greatest risk to develop alcohol and drug problems—those with a family history of abuse, or a developmental history of antisocial behavior, and those from ethnic and racial minority groups, for example—because many of these children may remain physically or psychologically beyond the reach of traditional, school-based prevention programs. Similar problems exist with college campus-based prevention programs, which have developed in increasing numbers in recent years. They have produced evidence for changes in the knowledge bases and attitudes concerning alcohol and drugs (*22*, *23*). However, they have not been able to document changes in drinking behavior in persons at greatest risk of misusing alcohol and drugs (*24*).

Control of availability. Unlike educational programs, laws and regulations designed to control the availability of alcohol, by making its acquisition more expensive or raising the minimum age of purchase, have had a demonstrable impact on both use and misuse.

Even relatively small increases in the price of distilled spirits (largely from State taxes) have been reported to reduce both consumption and deaths from cirrhosis and automobile crashes (*25*, *26*). Data from a related series of studies suggest that higher prices for beer, the alcoholic beverage of choice among youth, reduce both the number of young people who drink and the incidence of both heavy and frequent drinking (*27*, *28*).

Saffer and Grossman investigated relationships between State excise taxes on beer and motor vehicle accident mortality rates for young people during the years 1975–81 (*29*). They found that States with relatively high excise taxes on beer had lower death rates from motor vehicle accidents for those 15 to 24 years of age than States with lower excise taxes. Extrapolating from the findings, the investigators estimated that, had the Federal excise tax on beer risen with the rate of inflation since 1951 (it did not), the lives of 1,022 youths would have been saved.

In a related review, Ornstein and Levy concluded that, in this country, beer is relatively price-inelastic, distilled spirits is price-elastic, and the price-elasticity of wine remains uncertain (*30*). In other words, changes in the price of beer are more likely than those of spirits or wine to affect consumption, largely because beer is the alcoholic beverage of choice among the young, for whom price can be an important barrier to consumption.

The findings, taken together, encourage the view that restricting the availability of alcoholic beverages by making them more expensive impacts on both alcohol consumption and its consequences. In the United States, this impact appears to be greatest on younger people, for whom price is a more important issue.

The findings do not respond directly to a related question, however, that asks whether price is as important a determinant of consumption for those who habitually and regularly abuse alcohol as it is for those who do not. Most observers doubt whether price alone impacts substantially on rates of alcoholism in most western countries, because the continued availability of alcohol to those dependent on it is more important than its modest cost.

Increasing the minimum age of purchase does appear to have affected rates of alcohol misuse. Wagenaar reported reductions of 16 percent in single vehicle night-time crash involvement and 19 percent in police-reported, alcohol-related injuries after legislation raising the minimum drinking age to 21 in Michigan was passed (*31*). The effect of the nationwide drive by the National Highway Traffic Safety Administration to raise the minimum age of purchase was a net 13 percent fewer fatal crash involvements per year per affected driver in the 13 States where the minimum age of purchase had been raised (*32*).

However, there is a major difficulty in interpreting data on the impact of changes in minimum drinking age. At about the same time that efforts began to raise the minimum age of purchase, both State and local authorities as well as voluntary groups began campaigns to reduce the incidence of drunken driving by other means. The means included markedly increased enforcement efforts, a dramatic stiffening of the legal and financial consequences of drunken driving, more public attention and efforts by citizens' groups like MADD and SADD, and a jump in public education activities. The net effect, a significant reduction in the involvement of youth in alcohol-related crashes during most of the past several years, likely has been a function of all factors, those affecting availability and those focused directly on drunken driving.

While we have seen a reduction in alcohol-related crashes, we cannot be sure just how they came about, which makes it more difficult to undertake similar changes elsewhere.

Prevention summary. The survey of current approaches to prevention suggests that meaningful changes in consumption as a direct result of alcohol education programs have been difficult to document, especially for alcohol dependent persons.

Restrictions on the availability of alcohol, by raising taxes as well as the minimum age of purchase, have brought about changes in consumption by youths and in its consequences. Whether control of availability affects 20 percent of the population who consume 80 percent of the alcoholic beverages is unknown. The issue is debated by preventionists who support control of availability and those who oppose it. Control of availability, to the limited extent it has been used in this country, has not been shown to seriously impact on drinking by alcohol dependent persons.

Intervention

While treatment for those who have developed long-term alcohol dependence appears to yield modest positive benefits, predictors of positive outcomes relate more to the personal resources clients bring to treatment than to variations in the treatments themselves. Moreover, we are able to prevent some of the consequences of alcohol misuse, including drunken driving and the fetal alcohol syndrome, better than we are able to prevent long-term alcohol abuse itself.

Taking these conclusions as valid, greater efforts to intervene in developing patterns of alcohol misuse would make sense. Three promising efforts of this kind include early intervention programs for alcohol misusers who have not yet developed the physical and behavioral stigmata of chronic alcoholism; relapse-prevention programs, which recognize the difficulties for both the recovering alcoholic and the early abuser in maintaining long-term abstinence, which are different from the difficulties in achieving abstinence in the first place; and rational means to heighten the motivation of alcohol misusers and abusers to make meaningful changes in their drinking behavior.

Intervention with young adults. Many investigators have reported promising results from trials of treatment with a unique group of clients (*33–39*). Typically recruited through newspaper advertisements, clients in the trials were selected to ensure that they were in good physical health, did not have a history of psychiatric treatment, had maintained social stability and support, had been problem drinkers for a relatively short period of time, did not see themselves as alcoholic, rejected abstinence as a long-term treatment goal, and wished to receive brief treatment that would be minimally disruptive of their daily routine. While variously labelled, these clients were perhaps best considered to be young adult alcohol misusers. Few would have met *DSM–III–R* (Diagnostic and Statistical Manual of Mental Disorders, 3*rd* Ed., Revised. American Psychiatric Association, Washington, DC, 1987) criteria for moderate or severe alcohol dependence.

Typically, the clients were seen for several months in outpatient settings. The goal of their brief treatment, in virtually every instance, was to enable them to stabilize, and ultimately, to moderate their drinking, so that it did not reach alcoholic levels. A variety of behavioral techniques were employed to teach the clients self-control skills to help them make more appropriate choices about when, where, and how much they drink.

The overall results of attempts to induce young adult problem drinkers to modify a risky pattern of drinking have been promising. Typical reductions in rates of drinking of 30 percent and more have been reported, although followup periods have rarely extended beyond a year. Nonetheless, brief interventions intended specifically for persons who have just begun to misuse alcohol, designed to prevent the development of alcoholism in the future as much as to treat current misuse, hold considerable promise. Additional research on outcomes is needed in order to confirm the durability of the treatment effects, determine the identity of clients for whom this kind of intervention is appropriate, and investigate the extent to which the interventions actually inhibit the progression of drinking to alcoholic proportions.

The developers of broad-based employee assistance programs and wellness programs in industry have begun to target the same client population and to entertain similar goals. One of their aims, relatively new, is to attract to treatment not only those whose alcohol and drug problems are of long duration and great severity, but those who recently had been arrested for the first time for drunken driving, started to experience problems on the job or at home because of alcohol or drugs, or begun to ask themselves whether they were beginning to develop a problem. They are the individuals who do well in the less insistent interventions, those associated with wellness programs and generally focused on other health risks (*40*, *41*). While there is little data on the degree to which the programs actually succeed in inducing early alcohol and drug misusers to commit themselves to a program designed to modify their drinking pattern, the thrust of the growing effort is clear and data may be expected to result. (*42*, *43*).

Cognitive mediation of relapse-related phenomena. Marlatt and coworkers have described the importance of controlling cognitively mediated, relapse-related phenomena during the recovery and maintenance period of both alcohol dependence and alcohol abuse (*44*, *45*). Considering the post-treatment cognitions of recovering alcoholics, Marlatt and coworkers emphasize what they call the "abstinence violation effect," the scenario in which a single "slip" by the recovering alcoholic inexorably leads him or her to conclude that recovery is impossible, making a quick return to alcoholic drinking inevitable.

Marlatt and coworkers propose a series of cognitively based interventions to alter the alcoholic's conviction that a single "slip" condemns him or her to a lifetime of alcoholism, degradation, and worthlessness. They offer hope and a set of intervention approaches to those who share the belief that treatment does not end when the patient leaves the hospital and that intervention during the maintenance phase of treatment is a long-term enterprise that requires as much attention from both client and clinician as the inpatient treatment that precedes it.

More recently, Marlatt and his coworkers have applied cognitive therapy methods derived from his relapse-prevention model to heavy drinking University of Washington fraternity members (*46*). Recognizing the justified association between alcohol misuse and fraternity drinking practices, Marlatt developed an association with a fraternity at the university that permits him to test intervention strategies designed to help moderate drinking behavior.

Treatment motivation and outcomes. Miller (*47*) and coworkers (*48*) described the factors which influence motivation for treatment. Their aim was to develop procedures to heighten motivation, on the justified assumption that increasing motivation for treatment will increase the likelihood that alcohol-abusing clients will benefit from treatment.

Miller concluded from an extensive review of the literature that motivational programs need to provide the abusive drinker with feedback from a trusted friend or counselor about the likelihood of the drinker developing serious alcohol-related problems (*47*). Miller's conviction derives in part from a series of studies conducted in this country and in Europe that indicate that relatively brief interventions, properly performed, can have a lasting impact upon problem drinkers. The impact can be comparable to much more extensive, costly, and prolonged interventions (*49–51*). Miller and others are impressed with Edwards' classic demonstration (*52*) that brief "advice" about the seriousness of a drinking problem can, under certain circumstances, lead to changes in the drinking behavior of alcohol dependent persons, changes as pronounced as those induced by more extensive treatment.

At the core of Miller's effort to heighten motivation is the Drinker's Check-up, a 3-hour assessment procedure ostensibly designed to detect both risk factors and the harmful consequences of overdrinking on neuropsychological, physical, social, and psychological behavior (*53*). Implicit in the assessment is Miller's conviction that informing the early-stage problem drinker of the details of alcohol's harmful effects may lead the drinker to do something about the drinking, something the drinker might not otherwise do.

Initial research on the Drinker's Check-Up indicates that the results of this assessment have greater short- and long-term effects on drinking when the feedback is presented in such a way as to elicit and reflect the drinker's reactions to it, rather than in a confrontational manner. Miller explains these findings by pointing to the importance of inducing alcohol misusers to take personal responsibility for a change in their drinking behavior. The Drinker's Check-Up, and the manner in which its results are given the subjects, represent a promising vehicle for getting drinkers to take responsibility for their own behavior and to benefit from the brief but accurate advice the counselor is able to give.

Conclusion

Effective alternatives to traditional treatment approaches are available for long-term, alcohol dependent persons. New methods of prevention and innovative, brief interventions used in treating those less severely dependent, offer a wide array of tools with which to confront the alcohol and drug problems of patients.

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Genetics and Biological Markers of Risk for Alcoholism

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Synopsis . . . .

Substantial scientific evidence has accumulated that both genetic and environmental factors predispose the development of alcoholism in certain individuals. Evidence has accumulated to indicate that alcoholism is a heterogeneous entity arising from multiple etiologies. The demonstrated role of genetics in increasing the risk of alcoholism has promoted the search for biological markers that could objectively identify individuals who are genetically predisposed to alcoholism. Identifying such markers could allow for early diagnosis, focused prevention, and differential and type-specific treatment of alcoholism. Promising markers have been provided by research in electrophysiology, endocrinology, and biochemistry. Recent advances in molecular genetics are offering prospects for direct analysis of the human genome to determine elements that provide predisposition to, and protection from, alcoholism. Recent advances in research and new knowledge gained by the alcoholism treatment community and the lay public are helping to diminish the societal damage caused by alcohol abuse and alcoholism and to change prevailing attitudes about them.

THE DEBATE about whether ‘‘nature’’ or ‘‘nurture’’ is the prime determinant of risk for alcoholism continues to consume substantive energy and resources (for example, *Traynor and McKelvry* v. *Turnage*, argued in the Supreme Court of the United States) (*1*). However, behavioral genetics research long ago provided the means to examine the relative contributions of genetics and environment to behavioral differences among individuals (*2*). The application of these techniques to the study of human populations has demonstrated that both the environment (nurture) and inherited factors (genetics, or nature) play a role in predisposing a person to alcoholism.

In the 19th century, Sir Francis Galton studied the relative contribution of the environment and inherited factors in determining human behavior. His conclusions (*3*) that ‘‘nature prevails over nurture’’ when one studies a particular stratum of a population have met considerable resistance because these conclusions have, at times, been generalized indiscriminately to many questions on the determinants of behavior.

The formula $Vp = V_G + V_E$ states that the ‘‘phenotypic variance’’ (Vp), or the range of characteristics of individuals in a population, is the *sum* of the environmentally determined variance (V_E) in the characteristics and the genetically determined variance (V_G) in the characteristics (*2*). That is, both environment and genetics are involved. This formula does not indicate the relative contributions of environment and genetics in determining a particular phenotype—for instance, alcoholism. Relative contributions must be determined by experiment.

Familial Factors

The hypothesis that genetic factors have a role in alcoholism is based on observations that alcoholism runs in families. Cotton surveyed the literature on the families of 6,251 alcoholics and 4,083 families of nonalcoholics (*4*). The most striking finding of the survey was that ‘‘regardless of the nature of the population of non-alcoholics studied, an alcoholic is more likely than a non-alcoholic to have a mother, father or more distant relative who is an alcoholic.’’ A similar conclusion was reached by Goodwin (*5*) in an earlier review of family studies of alcoholism.

The literature on families of alcoholics also demonstrates the relatively consistent finding that other forms of mental illness such as schizophrenia and other major psychoses are not found at higher frequencies in families of alcoholics compared with the general population (*4, 5*). Although a number of studies have indicated that depression and psychopathological features are more frequently observed in relatives of alcoholics than in nonalcoholic families, the incidence of alcoholism was found to be higher than any other form of mental illness in families of alcoholics.

The available data on the families of alcoholics support the contention that alcoholism runs in families, but the data are not sufficient to conclude that genetic factors play a role in the etiology of alcoholism. The social system of the family may be an important and possible overriding determinant in the development of alcoholism. Kaufman (*6*), who summarized the family system variables that are important in the genesis of alcoholism, concluded that a search for a simple model of an alcoholic family is not realistic and that more complex interactive models are needed to describe the determinants of alcoholism. The needed interactive variable may be genetics.

Studies of identical (monozygotic, or MZ) and fraternal (dizygotic, or DZ) twins and adoption studies are needed to develop such models and to quantify the genetic and environmental contributions to individual differences in behavior. In one of the earliest completed studies, Kaij (*7*) examined 174 sets of twins and demonstrated that the concordance rate for alcoholism was 71 percent in MZ twins, but only 32 percent in DZ twins. A later study by Hrubec and Omenn (*8*) again demonstrated a significantly greater concordance for alcoholism in MZ twins than in DZ twins. In this study, 26 percent of MZ twins but only 12 percent of DZ twins were concordant for alcoholism. Although there remains some controversy in the area of twin studies of alcoholism (see, for example, an editorial by Gurling (*9*)), most studies support the hypothesis that genetic factors make a significant contribution to the etiology of alcoholism.

Studies of twins have been used to assess whether a person's physiologic (for example, electroencephalographic (EEG)) responses to ethanol, similarities in drinking patterns, and the metabolism of ethanol by a person are in part under genetic control (*10–12*). Such studies have further demonstrated that genetic factors play an important role in individual responses to alcohol, alcohol metabolism, and drinking patterns, as well as in the development of alcoholism. The contribution of environmental variables to producing similarities or differences between twins depends in great part on the measure being studied. Thus, MZ twins demonstrate almost identical EEG responses to alcohol, while the concordance values for drinking patterns and alcoholism in MZ twins indicate a substantive contribution of environmental variables, as well as genetics, to the twins' characteristics.

> *'The studies of the molecular genetics of alcohol and aldehyde metabolizing enzymes provide the first opportunities to use molecular probes of the human genome as markers of predisposing factors as well as protective factors in alcoholism.'*

The interaction of genetic and environmental factors in the development of alcoholism can also be studied by comparing individuals who have been adopted soon after birth with those who have been raised by their biologic parents. Such adoption studies have focused on the development of alcoholism in subjects whose biologic parents are alcoholics and who are raised by their natural parents or by adoptive families in which there is no alcoholism. These studies also have examined children born to nonalcoholic parents and raised in their own or foster nonalcoholic families. The most detailed and extensive adoption studies have been conducted by Cloninger and coworkers (*13*). These studies agree with the earlier work of Goodwin (*14*) and demonstrate that (*a*) adopted sons of alcoholic biologic parents are four times more likely to become alcoholic than adoptees whose biologic parents were not alcoholic; (*b*) sons of alcoholic biologic parents are more likely to be classified as alcoholic at an earlier age than their peers; and (*c*) daughters of alcoholic fathers, although not demonstrating a greater incidence of alcoholism, exhibit a high incidence of somatic anxiety and frequent disabling physical complaints.

Further analysis of the data collected by Bohman (*15*) demonstrated that the population of alcoholics being studied could be segregated into two prototypic groups. Both groups were characterized by the severity of their problems with alcoholism (for example, age of onset, violence associated with drinking, and attitude toward drinking) and, more recently, by personality traits (for example, novelty seeking) (*16*). The adoption studies are therefore notable not only for substantiating a genetic contribution to the development of alcoholism, but also

Heterogeneity of Alcoholism: Distinguishing Characteristics

Distinguishing features	Type 1 alcoholism	Type 2 alcoholism
Characteristics of biological parents:		
Father	Minor alcohol abuse Minor legal encounters	Severe alcohol abuse Serious legal encounters
Mother	Mild alcohol abuse Minor legal encounters	No alcohol abuse No legal encounters
Postnatal environmental characteristics		
	Determines frequency and severity of alcoholism in susceptible sons	No influence on frequency, but may influence severity
Severity of alcoholism		
	Usually isolated, less severe problems, but may be severe	Usually recurring, moderate to severe problems
Characteristics of alcohol-related problems:		
Usual age of onset	After 25 years	Before 25 years
Spontaneous alcohol seeking (inability to abstain)	Infrequent	Frequent
Characteristics of alcohol-related problems: (cont'd.)		
Fighting and arrests when drinking	Infrequent	Frequent
Physiological dependence (loss of control)	Frequent	Infrequent
Guilt and fear about alcohol dependence	Frequent	Infrequent
Personality characteristics:		
Novelty seeking	Low	High
Harm avoidance	High	Low
Reward dependence	High	Low
Relative risk in genetically predisposed sons		
	Two times, with postnatal provocation No difference without postnatal provocation	Nine times, regardless of postnatal environment

NOTE: Relative risk is the ratio of the risk of alcoholism in congenitally predisposed sons to the risk in others. A relative risk of 1 means there is no difference in risk of alcoholism between congenitally predisposed sons and others.
SOURCES: References *13*, *16*, and *54*.

for underscoring the fact that alcoholism, as defined by current psychiatric criteria (*17*), is a heterogeneous entity that probably involves a number of genetic as well as environmental factors.

A compilation of the distinguishing features of the two types of alcoholism described by Cloninger and coworkers (*13*) is shown in the box. Cloninger has cautioned (*16*), however, that these two subgroups of alcoholics may be "polar extremes" of a continuous variable rather than "discrete disease entities."

Family, twin, and adoption studies show that the risk for alcoholism is determined by genetic factors as well as by environment. The degree of genetic contribution depends on the subtype of alcoholism being examined and on the alcohol-use trait being studied. The conclusion that there is a significant genetic component to alcoholism, as is indicated, leads to the realization that the individuals who are at greater risk for becoming alcoholic because of inherited (genetic) factors are biologically different from individuals who have few or no inherited factors that predispose them to alcoholism.

Search for Markers

This realization sets the stage for the search for "markers" to identify the individuals at increased risk for alcoholism. The markers of increased risk, or predisposition, may be the actual elements that are mechanistically involved in the etiology of alcoholism, or they may be factors that in themselves do not predispose to alcoholism but are closely correlated with the development of alcoholism in an individual. Individual differences that can act as markers of a predisposition for alcoholism could be behavioral, physiologic, or biochemical. Given the array of possible markers, research has followed a pattern in which measurable differences between alcoholic and control subjects were initially considered as candidate markers. Clearly, differences between alcoholic and control subjects can be a result of years of alcohol consumption by the alcoholic (state markers) rather than differences associated with a predisposition to alcoholism (trait markers). Therefore, researchers have considered that a candidate trait marker either has to continue to be present during long periods of abstinence or should be present before the development of alcoholism in an individual. In certain instances, studies of animal models of alcoholism have been extremely informative in distinguishing state and trait markers.

Given the evidence that children of alcoholics are at greater risk for developing alcoholism (*4, 5, 7, 8, 10–14*), one would expect that the prevalence of a candidate trait marker would be significantly greater in a population of individuals who have a positive family history of alcoholism. More important, the candidate marker should predict, with a high degree of accuracy, the individuals who, when followed longitudinally, will develop alcoholism. A major caveat to be considered when assessing the utility of a candidate marker is that the probable heterogeneous nature of alcoholism may not allow for the generation of a single marker that can identify all individuals at risk for alcoholism. But even given the many difficulties and caveats, research on trait markers in alcoholism is providing some tantalizing insight into the future for the diagnosis of increased risk for alcoholism.

Physiological Markers

Measurement of brain electrical activity using scalp electrodes provides a noninvasive assessment of brain function. Two types of measurements have been commonly used in this regard. Electroencephalographic measures provide for the characterization and quantitation of the spontaneous electrical activity of the brain. Measures of evoked event-related potentials (ERPs) provide knowledge of the responses of the brain to external sensory stimuli. ERPs can be measured from a number of areas of the scalp after the delivery of a discrete sensory stimulus. Auditory and visual stimuli have been used most often in studies of alcoholics or of offspring of alcoholics.

The measurement of some of the later phases of ERPs has provided the most intriguing prospects for developing trait markers for increased risk for alcoholism. The P300 component of an ERP is a large positive deflection that occurs approximately 300–500 milliseconds after the presentation of a stimulus. The P300 component can be significantly enhanced in response to a relevant "target" stimulus presented within a series of frequently occurring nontarget stimuli. Porjesz and Begleiter (*18, 19*) found that P300 amplitudes were significantly diminished in alcoholics compared with control subjects when either relevant target stimuli or nontarget visual stimuli were presented. In a followup study of the alcoholic subjects during 4 months of abstinence, the subjects continued to manifest this "sensory-filtering" deficit.

The irreversible nature of the P300 deficits in alcoholics suggested that these deficits may be inherent characteristics of these subjects and may have been present prior to the onset of alcoholism. To test this possibility, Begleiter and coworkers (*20*) examined young (7–13 years old) sons of alcoholic fathers and control boys matched for age and socioeconomic status from families with no history of alcoholism. The experimental paradigm involved a complex visual task. When the ERPs were analyzed, the sons of alcoholic fathers were found to have a pattern of P300 response quite similar to the pattern found in abstinent alcoholics and significantly different from the P300 pattern found in sons from nonalcoholic families. Although some controversy still exists regarding the influence of task requirement (*21*) and the characteristics of the subject population (that is, sons of type 1 *versus* type 2 alcoholics) on the robustness of the witnessed deficits in P300 ERPs in sons of alcoholics, these measures have opened a fruitful area for the study of heritable markers of risk for alcoholism.

Electroencephalographic studies have also demonstrated long-lasting differences between alcoholic and control subjects. Particularly, alcoholics have been shown to manifest an excessive proportion of fast, or beta, EEG activity (*22*). Gabrielli and coworkers (*22*) also studied children of alcoholics and compared the results with those obtained from children of nonalcoholic parents. Interestingly, these investigators observed that male children of alcoholics demonstrated excessive fast EEG activity compared with control children. Further studies focusing on EEG responses to administered alcohol in young adult children of alcoholics (*23*) have demonstrated that individual responses to alcohol in terms of EEG activity may be influenced by whether or not an individual is a son of an alcoholic father. The electrophysiologic studies of offspring of alcoholics, in total, provide a solid foundation for the search for markers of predisposition to alcoholism and a foundation for the search for etiological mechanisms for alcoholism.

Subjective measures of the response to ethanol have also been investigated in an attempt to identify a genetically linked trait marker for a predisposition to alcoholism. It has been reported that individuals with an alcoholic first-degree relative—that is, family history positive (FHP) subjects—showed a decreased response to ethanol compared with matched family history negative (FHN) subjects. To measure the subjective responses to consumed alcohol, subjects completed a questionnaire measuring aspects of intoxication ranging from pleasant feelings to feelings of discomfort (*24*). In these studies, there was no difference in the baseline mood (before ethanol administration or during placebo administration) between the two groups.

More objective measures of the response to ethanol have also been examined in FHP and FHN individuals. Static ataxia, or body sway, a measure of psychomotor performance, has been used to evaluate baseline differences between children of alcoholics and children of nonalcoholics. Static ataxia also has been used to measure differences in the response to ethanol of these groups. Measurement of static ataxia involves placing a harness around the upper body and asking subjects to stand still with their arms and feet in a particular position (usually with their arms folded across the chest or at their sides and with their feet close together) and with their eyes open or closed. The harness is attached to cords which are connected to sensors that measure lateral or anterior-posterior movement. A movement platform method has also been used to assess body sway.

In several studies, it has been found that FHP individuals show greater baseline body sway than FHN individuals (*25, 26*). The ability of a subject to control body sway at a given blood ethanol level has been suggested as a measure of ethanol tolerance (*25*). When body sway was measured after consumption of ethanol, it was found to be less in FHP than in FHN individuals. This was observed even when the subjects were matched for drinking history and were assessed at the same blood ethanol levels and even when heavy drinkers were excluded (that is, the effect of tolerance was theoretically minimized) (*27*). In this particular study, in contrast to earlier studies, FHP and FHN subjects were not found to differ in baseline (during placebo administration) body sway. However, the data are consistent with the hypothesis that those with a positive family history of alcoholism have a decreased psychomotor response to ethanol. Nonetheless, the possible influences of ethanol tolerance, the baseline differences between the groups, and the importance of the subtype of alcoholism being studied, need further investigation.

Endocrinological responses to ethanol in FHP and FHN individuals also have been examined. Plasma levels of cortisol and prolactin increase after ethanol is ingested, and differences in these responses, have been reported between FHP and FHN men. FHP and FHN subjects in one study, after ingesting a 0.75 ml per kg dose of ethanol, showed similar initial increases in prolactin, but prolactin levels dropped more rapidly in the FHP subjects than in the FHN subjects (*28*). This finding was replicated by using a design that incorporated a placebo and two ethanol doses (*29*). Similarly, FHP men in another study showed a decreased cortisol response after ethanol ingestion (*30*). As in the earlier study, subjects were carefully matched for drinking history. Consequently, their decreased response to ethanol may well be an indicator of a predisposition to alcoholism. The possible influence of prior ethanol consumption and the cortisol and prolactin responses to ethanol in defined subtypes of alcoholics, however, are factors that need further evaluation. This latter distinction is particularly important when one considers that the subjects in most of these studies are college students, and this population is not likely to include a large proportion of type 2 alcoholics.

As mentioned above, it seems likely that no one marker will be adequate to identify all individuals at increased risk for becoming alcoholic. It has been demonstrated that measurement of a combination of variables provides a better discrimination of FHP and FHN individuals (*31*). The most realistic goal at present would appear to be identification of the fewest number of variables that will provide the maximum specificity and sensitivity for classifying subjects at risk for developing alcoholism.

Biochemical Markers

Biochemical markers of a predisposition to alcoholism would consist of gene products, or closely linked gene products, that predispose an individual to ethanol-related problems. It has often been assumed that a biochemical marker for a predisposition to alcoholism should be present prior to alcohol consumption. Such a marker possibly could be a biochemical variable that is altered differently by alcohol in individuals who are predisposed to alcoholism, compared with individuals who have no predisposition.

The enzyme monoamine oxidase (MAO) has been suggested as a possible marker for alcoholism or a predisposition to alcoholism. MAO is a mitochondrial enzyme involved in the metabolism of biogenic amines. There are two forms of MAO, each having different substrate specificities and inhibitor sensitivities as well as different tissue distributions. The brain, for example, contains both MAO A and MAO B, while the B form is found in human platelets. Platelet MAO activity is under genetic control (*32*), and low platelet MAO activity has been suggested as a marker for schizophrenia and several other psychiatric disorders, as well as for alcoholism.

In at least a dozen studies, mean platelet MAO activity has been reported to be significantly lower in alcoholics than in controls (*33–35*). Included are studies that evaluate from approximately 20 to 100 active or abstinent alcoholics and matched controls. However, substantial variability in MAO activity has been noted within alcoholic and control populations, leading to significant overlapping in activity between these groups. There are a number of possible explanations for such variability. A recent study, for example, demonstrated that the maximum velocity (V_{max}) for platelet MAO, but not the affinity (K_m) for substrate, was altered in alcoholics, compared with controls (*34*). If a single low substrate concentration is used to measure MAO activity, variability may be more prevalent.

As for all potential markers, particularly those measured in alcoholics, it is necessary to determine whether the observed difference is an effect of alcohol consumption or whether it represents an inherent characteristic of the individuals. In animal studies, little change in brain MAO activity was evident when mice or rats were fed ethanol chronically (*36, 37*), suggesting that MAO activity is not easily altered by prior consumption of ethanol. However, platelet MAO activity in alcoholics was found, in several studies, to fluctuate after cessation of alcohol intake (*38, 39*). In part, these fluctuations may be due to changes in platelet production that have been documented during intoxication and withdrawal in some alcoholics, because newly matured and released platelets have high levels of MAO activity. Such possible additional sources of variability emphasize the difficulties of evaluating genetic markers in alcoholics and also demonstrate the value of using populations such as twins or FHP and FHN individuals. In a study involving a small group of FHP and FHN men, there was a trend toward lower platelet MAO activity in the group with alcoholic relatives (*35*). In a more recent study, when alcoholic subjects were divided into type 1 and type 2 subtypes, it was found that type 2 alcoholics had significantly lower platelet MAO activity than controls, while the activity in type 1 alcoholics was similar to that in controls (*40*). In another study, it was reported that teenage boys who were abusers of multiple drugs and who exhibited personality traits consistent with type 2 alcoholism had low platelet MAO activity (*41*).

These data suggest that much of the variability and overlapping observed both in studies of alcoholics and control subjects and of FHP and FHN men may arise from the failure to distinguish subtypes of alcoholism. In spite of the variability, the relatively consistent finding of low platelet MAO, particularly in those individuals with a highly heritable form of alcoholism, suggests that MAO enzyme activity may be useful as a trait marker for alcoholism. The specificity of this marker to alcoholism remains to be evaluated, however, because, as mentioned earlier, platelet MAO activity may also be low in individuals with schizophrenia and other mental disorders.

Another characteristic of MAO that might be useful in distinguishing alcoholics and controls is the susceptibility of the B form of the enzyme to inhibition by ethanol in vitro. In a group of 53 alcoholics and 17 controls, matched for age, race, and socioeconomic status, inhibition of platelet MAO activity by ethanol was found to be significantly greater in alcoholics than controls when a high substrate concentration was used to assay MAO activity (*33*). The difference was not associated with race, smoking, or illicit drug use, and there was no significant correlation with age, duration of problems with alcohol, or time since the last alcoholic drink. The latter data suggest that the difference in the properties of MAO is not a reflection of chronic ethanol consumption, but may be an inherent characteristic of alcoholics.

Adenylate cyclase (AC) is another enzyme localized in platelets and lymphocytes that has recently been investigated in alcoholic subjects. This enzyme is activated by hormones and neurotransmitters and catalyzes the conversion of adenosine triphosphate (ATP) to the ubiquitous intracellular "second messenger," cyclic 3′,5′-adenosine monophosphate (cyclic AMP). In a study of 94 male alcoholics and 31 matched controls, basal (unstimulated) platelet AC activity was the same, but stimulation of AC by fluoride, guanine nucleotides, and prostaglandin E_1 was significantly lower in the platelets of alcoholics than in the platelets of controls (*33*). As for the inhibition of MAO activity by ethanol, lower fluoride-stimulated platelet AC activity was not associated with smoking, race, or illicit drug use and was not significantly correlated with the duration of problems with alcohol or time since the last consumption of alcohol. In fact, in this study, platelet AC activity was lowest in alcoholic individuals who had reportedly abstained from alcohol for 1–4 years. The data suggest that low platelet AC activity could be a genetically influenced characteristic of alcoholic subjects. Discriminant analysis, using the values for ethanol-inhibited MAO activity and fluoride-stimulated AC activity, was able to provide correct classification of 75 percent of alcoholics and 73 percent of controls.

A more recent analysis of platelet AC activity involved a group of Swedish male alcoholics and controls. The results indicated that fluoride- and guanine nucleotide-stimulated AC activities were again significantly lower in platelets of alcoholics than in platelets of controls, and a significant *negative* correlation was found between fluoride-stimulated AC activity and time since the last consumption of alcohol. These findings again suggest that lowered platelet AC activity is not a reversible effect produced by chronic ethanol consumption. Furthermore, in these individuals, there was a significant effect of hereditary factors on platelet AC activity, in that activity was lowest in individuals who had a higher number of alcoholic first-degree relatives (*42*). It has also been reported that in Japanese alcoholics, *in vitro* stimulation of platelet AC activity by ethanol was lower than in controls, and this difference was not reversed at 4 weeks after withdrawal (*43*).

Overall, the data suggest that platelet AC characteristics may represent a trait marker for predisposition to alcoholism. The actual site of this marker may be G_s, the guanine nucleotide-binding protein that couples receptors to AC, because stimulation of AC by a number of agents acting via G_s is reduced in platelets of alcoholics. It has also been reported that adenosine-stimulated AC activity is lower in *lymphocytes* of alcoholics compared with controls (*44*), and there is a preliminary report of decreased guanine nucleotide-stimulated AC activity in lymphocytes of alcoholics (*41*). When lymphocytes from alcoholics were grown, *ex vivo*, in culture, they recovered their responsiveness to adenosine and had higher cyclic AMP levels than cells from controls. However, the cultured lymphocytes from alcoholics continued to be much more sensitive to the effect of ethanol in vitro than lymphocytes from control individuals (*46*). Studies with lymphocytes have, therefore, provided further evidence that differences in AC activity may represent an inherent characteristic of the cells of alcoholic subjects.

In addition to the study of specific enzyme activities that may reflect a predisposition to alcoholism, some attempts have been made to link the predisposition to alcoholism with other characteristics that are known to be inherited. For example, a significantly higher phenotype frequency of HLA antigen (human leukocyte antigen) CW3 was found in chronic alcoholics than in controls; this difference became even more distinct when alcoholics were subdivided according to whether they had liver disease (the CW3 phenotype frequency was higher in individuals with liver disease) (*47*). HLA antigens are proteins on the surface of cells that identify a tissue, and they are coded for by genes on chromosome 6. Another type of linkage was suggested in a study of 11 serological markers in alcoholics. In the original study of alcoholics and their nonalcoholic first-degree relatives, associations were reported between alcoholism and two blood groups, as well as a serum protein (*48*). A more recent preliminary report on 30 families in which alcoholism was segregated also included a reanalysis of the earlier data and revealed a linkage with the MNS blood marker, which is coded for by a gene on chromosome 4. Another locus (GC) on chromosome 4 also gave some evidence of linkage (*44*).

Such linkage studies require a careful choice of subjects and are still in the early stages. However, in combination with other types of research, they may eventually provide important information on the locus of genetic material associated with a predisposition to alcoholism.

Although the bulk of this review has centered on markers for predisposition to alcoholism, it has become clear over the past several years that the development of alcoholism involves an interplay between positively predisposing factors and factors that may produce an aversion to alcohol and thus protect an individual from becoming an alcoholic. An example of such a protective factor may be the inherited inability of some individuals to metabolize acetaldehyde, which is produced from ingested alcohol. The adverse physiologic responses of these individuals to the elevated levels of acetaldehyde in the circulation may act to diminish alcohol consumption.

A variant in human liver aldehyde dehydrogenase was described in the early part of this decade (*50*). More recently, molecular genetic studies have demonstrated a structural mutation in certain individuals in the gene coding for a human aldehyde dehydrogenase that has a high affinity for acetaldehyde (*51*). This structural mutation leads to a loss of the enzyme's ability to oxidize acetaldehyde. Population studies (*52*) have shown that the mutant enzyme and the diminished ability to metabolize acetaldehyde are prevalent among the Mongoloid populations (for example, approximately 44 percent of the Japanese exhibit the aldehyde dehydrogenase deficiency). The increased levels of circulating acetaldehyde after ethanol ingestion in subjects with an aldehyde dehydrogenase deficiency and the resultant flushing and other noxious consequences of acetaldehyde toxicity may be determinants of a lower incidence of alcoholism among Orientals. Goedde and Agarwal (*53*) have indicated that the frequency of the deficient aldehyde dehydrogenase phenotype in alcoholics in treatment programs in Japan is much lower than the frequency of this phenotype in the normal population or in patients in treatment for other mental illness. The studies of the molecular genetics of alcohol and aldehyde metabolizing enzymes provide the first opportunities to use molecular probes of the human genome as markers of predisposing factors as well as protective factors in alcoholism.

The scientific investigation of markers for ascertaining the risk of developing alcoholism is growing rapidly. Behavioral, physiologic, biochemical, and molecular genetic indicators of predisposition are being developed. The advances in this area have a multitude of implications beyond the obvious utility of being able to identify more accurately people at risk. The demonstration of the presence of biologic markers of predisposition should clearly promote the acceptance of the role of biology and genetics in the development of alcoholism. The availability of such markers should allow for swifter progress in elucidating the etiology of various forms of alcoholism. Differential diagnosis with the use of markers can provide for specifically focused treatment programs and circumscribed and directed prevention efforts.

The beneficial aspects of research on markers for predisposition to alcoholism, however, have to be matched by progress in a number of related areas for the full potential of this work to be realized. Destigmatization of alcoholism is a necessity, and the conceptualization of alcoholism as a moral weakness should no longer be accepted. Research on effective treatment and prevention modalities needs to be accelerated.

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A Biobehavioral Research Perspective on Alcohol Abuse and Alcoholism

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Synopsis

An empirical biobehavioral research approach to the conditions generally identified as alcohol abuse and alcoholism emphasizes the temporal ordering of participating biochemical, physiological, and behavioral events that provide an operational basis for characterizing the functional aspects of this complex disorder and identifying distinguishable features of the alcohol abuse and dependence process. The available evidence suggests that alcoholism is a condition determined by a host of continuous variables rather than an entity possessing static qualities that imply intractability. The challenge for biobehavioral research is to determine the details of how chronic and excessive alcohol drinking is generated as well as the conditions under which such overindulgence can be attenuated and prevented. Environmental context, for example, can dramatically alter the frequency and amount of alcohol intake. Such contextual malleability is suggested as an important key to at least some of the inconsistencies in the literature with regard to the conditions under which chronic and excessive alcohol intake occurs. Excessive and chronic alcohol ingestion would seem most parsimoniously viewed as a set of behaviors for which others might have been substituted, and intermittently do, rather than as a highly specific disorder or disease. Though current etiological, preventive, and therapeutic orientations emphasize the role of physical dependence and favor genetic influences as strong determinants of alcohol-related disorders, it is important to recognize that troubled drinking is malleable, waxing and then entering periods of remission, with alcohol drinking even in severely dependent individuals remaining susceptible to control by both antecedent and consequating environmental events.

IT IS THE PURPOSE of this article to provide a brief overview of the progress and prospects in developing biobehavioral research models of excessive alcohol ingestion and physiological dependence on ethanol. With regard to therapeutic and preventive measures, the modest insights gained from such biobehavioral research may help to answer perplexing questions that have been difficult to address directly in clinical settings. The thrust of this paper, then, is to indicate what laboratory biobehavioral research has to tell us about excessive alcohol ingestion and physiological dependence that may have new implications for the human conditions that are generally identified as alcohol abuse and alcoholism.

Basic Concepts and Definitions

Some definitional clarity has been gained recently in the analysis of drug abuse and dependence (of which alcohol problems must be regarded as a specific case) by dividing the vast arrray of events that characterize this area in general into two reasonably exclusive categories based upon explicit operational criteria (*1*). Such a division is possible by distinguishing events that occur *before* from events that occur *after* the actual intake of the substance. The defining operations of the ''before'' class would include (but are not necessarily limited to) proactive alcohol seeking and drinking behaviors associated with the consequences of excessive alcohol ingestion. The defining operations of the ''after'' class would focus upon the reactive biochemical, physiological, and behavioral changes associated with the consequences of excessive alcohol ingestion, as well as with the tolerance and abstinence effects after alcohol withdrawal following recurrent excessive alcohol ingestion. The temporal ordering of biochemical, physiological, and behavioral changes in relation to alcohol intake can thus provide an operational basis for analyzing the range of alcohol's functional characteristics and for identifying distinguishable features of its spectrum of action.

'Those who deal therapeutically with alcohol problems know the complexity of etiologic and maintaining factors as well as the web of social myths that surround the condition. These myths and expectations exacerbate clinical problems by perpetuating notions and explanations of alcoholism that endow terms such as ''addiction,'' ''loss of control,'' and ''abstinence avoidance'' with undeserved explanatory powers.'

The relevance and importance of this distinction between proactive ''alcohol abuse'' and reactive ''alcohol dependence,'' that is, ''alcoholism,'' resides in the fact that their defining properties are not coextensive, they do not invariably occur together, and the methods differ by which they are analyzed experimentally. The strength of proactive alcohol-seeking behaviors and abusive self-administration of alcohol can be maintained by use patterns with doses of alcohol that produce no significant tolerance or withdrawal (*2*, *3*). Conversely, tolerance and abstinence syndromes can be demonstrated under conditions that involve neither alcohol-seeking behaviors nor abusive alcohol self-administration (*4*).

Interactions between these proactive and reactive events of the alcohol scene are, of course, commonplace. Changes in alcohol-seeking and alcohol drinking can occur as sequelae to both the acute effects of alcohol and to the tolerance and withdrawal effects that follow more chronic and excessive alcohol ingestion (*5*). Conversely, the chemical and physiological changes that define ''physical'' dependence can as well be sequelae to the repeated excessive self-administration of alcohol (*6*). But the relative contributions of these distinguishable processes to alcohol-related problems can vary widely as a function of dose, environmental circumstances, and previous experience, including history of alcohol use (*7*).

This conceptual framework for encompassing alcohol abuse and ''alcoholism'' provides an empirical approach to clarifying the semantic and taxonomic confusion perpetuated by the interchangeable use of terms like ''addiction'' and ''dependence'' as referents for a bewildering range of phenomena and experiential pseudo phenomena (*8*). The terms themselves, persistently reified as substantive noun ''things'' in subject predicate relationships with other ''things'' (''causing'' as well as being ''caused by'' these other ''things''), are seldom accorded appropriate conceptual status as constructs emerging from observed interactions between specifiable antecedents (biological and social) and definable consequences (biochemical, physiological, and behavioral). Within this context, biobehavioral research can provide a basis for defining these constructs more operationally and for specifying the conditions under which a unifying conceptual framework can be developed for treating and preventing the prominent health hazards involved.

Biobehavioral Perspective

The proactive events associated with alcohol seeking and excessive alcohol ingestion are of primary interest and concern from a biobehavioral perspective. Those who deal therapeutically with alcohol problems know the complexity of etiologic and maintaining factors as well as the web of social myths that surround the condition. These myths and expectations exacerbate clinical problems by perpetuating notions and explanations of alcoholism that endow terms such as ''addiction,'' ''loss-of-control,'' and ''abstinence avoidance'' with undeserved explanatory powers (*9–13*). One advantage of laboratory research models is that animal behavior is culture-free in that it is not directly determined by human cultural prescriptions. It is relatively culture-free, that is, unless investigators read into models the results that bits of cultural mythology call for.

Animal model delineation should not be merely a ritual behavior of scientists to confirm culturally based beliefs about the nature of ''alcoholism.'' If that is what is chosen, then a model has little information to yield. One would have already decided what ''alcoholism'' is, and the models would furnish only an acceptable biomedical context in which to couch these beliefs so that we could get on with the business of re-stating what we already held to be the case: perhaps that alcoholism is a genetically determined behavior that specially and compulsively locks on to the ethanol molecule, primarily for reasons of individually flawed biochemistry, and inevitably leads to loss-of-control drinking for which the only cure is total and permanent abstinence. If we were certain of all these facts, then we would hardly need the models, for they would only produce tedious and expensive demonstrations. The purpose of a model is not to confirm our beliefs about how something works, but rather to find out if our notions are tenable, and if so, what further consequences flow from the model.

Construction of a laboratory animal model would be facilitated if there were general agreement as to the definition of a case of human "alcoholism." "The main criteria of alcoholism seem to be present in populations as continuous distributions rather than as discontinuous, isolatable entities (*14*). The bulk of the evidence suggests that the more appropriate analogy might be with hypertension rather than with coronary thrombosis or with osteoporosis rather than with pathological fracture" (*14*). The lesson to be drawn is that we will probably fare better by clarifying the variables that institute and maintain ethanol overindulgence and the range of behavioral antecedents and biomedical consequences than in trying to produce a miniature version of the definitionally vague "alcoholic." The strategy of producing several kinds of renovascular hypertension in animals has unravelled many of the variables that produce and maintain this condition, even though none of the animal laboratory models are entirely similar to human renovascular hypertension.

If we approach "alcoholism" as an entity possessing static qualities, implying an essentially intractable status, then we naturally expect to mimic this in animal models that are deemed successful. But no model has demonstrated anything like a compulsively "hooked" alcoholic animal with loss of control. The existence of such an unremittingly static entity in humans has been questioned on several grounds: the alcohol consumption of a population is distributed unimodally (not bimodally); troubled drinking is malleable, waxing and then entering periods of remission; drinking, even in severe alcohol dependence, remains susceptible to control by both antecedent and consequating environmental events (*8*, *15–17*).

It is time to see if our experimental arrangements uphold commonly received notions of what sustains overindulgence or, what is more likely, if we will be induced to revise some of our opinions. If "alcoholism" is like hypertension, then it is a condition determined by the interaction of a host of continuous variables, and we need to determine the details of how excessive drinking is generated, as well as what useful attenuators can be applied for therapeutic and preventive ends.

Experimental Findings

There is now a substantial experimental literature confirming that laboratory animals, including rodents and primates, will self-administer alcohol both intravenously and orally (*3*, *18–20*). The conditions under which such self-administration can become chronic and excessive, however, are not simply derivable from the pharmacological properties of ethanol. Unlike cocaine, for example, which dramatically preempts the stream of behavior over a range of experimental conditions, alcohol self-administration (particularly via the oral route) has been difficult to establish in the animal laboratory without special induction procedures. Perhaps noxious taste factors are involved (as they certainly are in some humans, including most children) or the slow onset of pharmacological action by the oral route, but chronic alcohol overindulgence has rarely been modeled in laboratory animals. But then, most people do not overindulge chronically either, and the challenge of determining the conditions under which such excessive alcohol ingestion can be observed enhances the relevance of an experimental analysis. A promising lead in this regard is suggested by the experimental evidence that the environmental context in which drug-taking occurs can dramatically alter the reinforcing function of even the most abusable substances. This contextual malleability of a drug's reinforcing efficacy has recently been documented with both cocaine (*21*) and nicotine (*22*), revealing that both drugs can have either pronounced reinforcing or punishing effects depending upon environmental contingency conditions. Biobehavioral research findings strongly suggest that such contextual malleability may hold the key to some of the apparent inconsistencies in the experimental literature with regard to the conditions under which chronic and excessive alcohol intake may occur.

The conditions for producing an explosive increase in oral fluid intake with laboratory animals, for example, turn out to be not all that complex. A relatively minor constraint on the availability of some important commodity (for example, food) and an intermittent schedule of access to that commodity is sufficient. Although never deprived of water, animals receiving food pellets on the average of once every minute drank 10 times as much water in 3 hours as they did when receiving the same number of pellets all at once (*23*, *24*). They drank about half their body weight in 3 hours when on such an intermittent food schedule. This overindulgence, which continues for months during daily intermittent feeding sessions, cannot be explained by any standard physiological, nutritional, or behavioral considerations (*25*).

This intermittent delivery of food has been demonstrated to increase the intake of a number of drugs taken orally in addition to alcohol: barbiturates, opiates, phencyclidine, and amphetamine (*26–30*). And excessive heroin, methadone, cannabis, and nicotine intake has been reported intravenously under such schedule-induced conditions (*31–33*). Alcohol was drunk excessively by a group of animals exposed continuously to an intermittent feeding schedule (*26*). The alcohol was preferred to water and other solutions, and chronically excessive intake under such intermittent feeding conditions resulted in severe physiological dependence (that is, abstinence syndrome following alcohol withdrawal). This model of excessive alcohol intake has been used to investigate several problems and consequences related to ''alcoholism.'' Among these are tolerance, cross-tolerance, cross-abuse, physical dependence, motor function, ethanol elimination rates, water-electrolyte status, liver pathologic changes, brain changes in adult, and body weight reduction in fetally exposed animals (*26*, *34–43*).

The excessive alcohol intake demonstrated in these experiments is in concordance with other kinds of excessive behavior that can be generated by the environmental context that determines schedule-induced behavior. For example, intermittent schedules of reinforcement for delivering food (or other commodities) can induce many kinds of excessive behavioral adjuncts including attack, pica, hyperactivity, inappropriate escape, elevated drug intake, and smoking (*44–50*). In general terms, while deprivation acts as a crucial facilitating condition, it is the episodic delivery of the valued, deprival commodity (food) in one domain that induces excessive behavior adjunctive to that domain. If we consider that both nature and society often provide an uneven flow of crucial commodities important to survival—food, territory, sex, social interactions—which all too often amounts to deprivation, then this can constitute intermittent scheduling with the possibility of generating excessive behavior.

The environmental conditions that give rise to schedule-induced behavior can apparently induce a variety of excesses, some productive (creative endeavors, workaholic overcommitment) and others characterized by social disturbances, violence, and drug abuse. The particular directions taken appear to depend upon current environmental opportunities and the individual's capacity and history to use an available commodity or activity. Environmental conditions that produce alcoholism can thus be seen as continuous with those producing other sorts of excessive behaviors and woes. Certainly, the diagnostically relevant determinants of alcoholism (degree of ethanol consumption and impaired job performance or social behaviors, or both) present as continuous population variables rather than as special and discrete disease entities. These considerations suggest that excessive and chronic alcohol ingestion represents a set of behaviors for which others might have substituted (and intermittently do), rather than as a highly specific disorder or disease state.

One characteristic of alcoholic drinking that should be considered by a research model is the volatility of the intake pattern. Chronic excess may be followed by relative moderation; bingeing may be succeeded by months of sobriety, only to slip into relapse. In human beings, it is not always clear why these changes occur, but an experimental model that can clarify the variables that turn on and off chronic excesses could illuminate volatility. By simple manipulation of the variables that produce and cut short schedule-induced excess, it has been easy to evoke such volatility (*51*, *52*). But even in experiments where the external circumstances are held constant day-to-day, episodes of self-imposed abstinence have been noted in monkeys (*53*) and humans (*54*).

A venerable theory of ''alcoholism'' is that the development of a state of physical dependence is a major factor in that it explains chronically maintained excessive intake. Inasfar as schedule-induction is the only voluntary (unforced) oral ingestion model that produces physical dependence so severe that death ensues from withdrawal convulsions, it is an arrangement that has been of use in evaluating this notion. It should be clear that physical dependence cannot account for the genesis of overindulgence. Frank physical dependence in humans is an end-stage phenomenon that is present only after some years of severe overindulgence (*54*).

The role of physical dependence in alcohol self-administration has been extensively reviewed (*55*, *56*), and it is now clear that the extraordinary focus on this aspect of the problem should perhaps be regarded as vestigial. Advances in an understanding of the factors that maintain self-administration have long since relegated physical dependence to a supporting role, albeit of potential significance. It remains a strong hypothesis, if not an act of faith, that physical dependence plays a central role in the maintenance of the self-administration of alcohol. While emphasizing the efficacy of the schedule-induction method in the production of physical dependence on ethanol, it must be recognized that this attests only to the robust intake level and its long-term maintenance. The resulting dependence state may just be unequivocal "proof of the pudding" and not a systematically crucial endpoint for the analysis of alcohol overindulgence. No clear evidence exists that chronic, excessive intake is maintained by avoidance of the withdrawal syndrome associated with physiological dependence.

The conditions sufficient for the induction of chronic ethanol overindulgence can also give rise to a host of other adjunctive, exaggerated behaviors, depending upon the context. This places alcoholism within a general framework of other excessive and pathologic behaviors that are primarily environmentally driven. By manipulating a few situational variables, the overindulgence can be turned on, off, or otherwise modulated, perhaps aiding in the analysis of the volatility of the alcoholic drinking-pattern. The food-limitation condition of this model is viewed as homologous to other facilitating variables that act in human situations to transform an agent of only moderate abuse potential into a more powerful one. Finally, schedule-induced overindulging in animals remains under the control of the environmental events that determine the excessive intake, even after a long history of overdrinking. The eventual physical dependence (while it can play a role in fluid preference) does not indicate that ethanol captures the ingestive behavior motivationally to transcend the original inducing conditions.

Therapeutic Implications

The very fact that long-term ethanol overindulgence in laboratory experimental models can be cut short by very simple changes in the scheduling of environmental events sounds an optimistic note with respect to therapeutic intervention. This dramatic reversibility clearly indicates that an overindulgence pattern is by no means self-perpetuating, even though the rest of the drinking context remains unaltered. Some interventions may only require appropriate alternatives to ethanol. Even in the presence of severe dependence, for example, with schedule-induced animals having had a long history of choosing ethanol in preference to water, availability of an oral glucose or saccharin solution that is concentrated enough alleviates the alcohol overindulgence.

The high levels of voluntary ethanol intake as a result of schedule-induction have been used to evaluate the ethanol intake-blocking properties of a few pharmacological agents. Disulfiram can decrease the intake of ethanol, mainly through the inhibition of aldehyde dehydrogenase. Both disulfiram and EMD 15,700 (also an aldehyde dehydrogenase inhibitor) markedly decrease the 5 percent ethanol intake of animals chronically exposed to schedule-induction, but they have little effect on animals that have water to drink rather than ethanol (*57*). In spite of repeated drug injections, however, the attenuation of ethanol intake does not appear to outlast the pharmacological action of the blocker. Upon drug discontinuance, the animals quickly resume drinking the 5 percent ethanol solution at the usual rate. In other studies (*58*), the specificity of both disulfiram and calcium cyanamide in blocking schedule-induced ethanol overindulgence in a dose-dependent manner has been confirmed.

Recently, the imidazobenzodiazepine Ro 15–4513 has been reported to block the anticonflict activity of low doses of ethanol, as well as the behavioral intoxication observed with larger ethanol doses (*59*). Since concern has been expressed that if the drug attenuated drunkenness it could encourage, rather than discourage, overindulgence (*60*), studies have been undertaken to evaluate its relative effects on both ethanol and water schedule-induced overindulgence. The drug decreased the intakes of both the ethanol and water groups equivalently, indicating perhaps that there is little danger that Ro 15–4513 will trigger uncontrolled bingeing.

Preventive Implications

Using the laboratory experimental models of excessive and chronic ingestion of alcohol to evaluate possible prevention strategies represents an important research challenge since the overindulgence can be so predictable, severe, and durable. Virtually all animals exposed to the experimental conditions develop chronic and excessive alcohol ingestion, and they belong to normal, unselected populations with presumably no bias toward the development of alcoholism. In human beings, as well, prevention efforts are aimed at populations thought to be at hazard for the development of alcoholism, but owing to genetic as well as environmental factors. The problem amounts almost to the attempted block of a fated identity. Alcoholism is one of the few diseases which one "becomes," suggesting a behavioral etiology. In our culture we

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"have" heart attacks and are "victims" of cancer, but "become" neither of these diseases (*61*).

While alcohol overindulgence is a highly probable response to the appropriate experimental conditions, the question does arise as to whether its development can be blocked by some other adjunctive behavior. In a recent study using the schedule induction model, a running wheel was present for the first 3 months as an adjunctive behavior alternative. When water was then made concurrently available, overdrinking was not only retarded, but upon a change to 5 percent ethanol *overindulgence failed to develop*. Animals drank only at known control levels. McMillan (*62*) found that when either water or 5 percent ethanol solution was available, making a running wheel available had either no effect or a minor one on schedule-induced drinking. However, when a choice between the two fluids was available (5 percent ethanol was much preferred over water), running-wheel availability decreased consumption of 5 percent ethanol and increased water consumption. It would appear that an alternative activity other than an ingestive one can interfere with established ethanol overindulgence. Adding the availability of a 0.9 percent NaCl solution alternative to an established 3-hour schedule-induced 5 percent ethanol overindulgence also results in a stable, marked reduction in daily ethanol ingestion (*63*). Of even greater interest is the finding that a history of schedule-induced 0.9 percent NaCl solution overindulgence greatly interfered with attaining later 5 percent ethanol overindulgence. Much of this latter research begins to suggest that such laboratory models of ethanol overindulgence at least might be dependent on the initial conditions of the experiment.

Two groups of animals, for example, were given differential fluid polydipsia histories (*64*). One had a history of choosing between 5 percent ethanol and 0.7 percent glucose (dilute, mildly acceptable), in standard daily 3-hour schedule-induction sessions, while the other similarly treated group chose between 5 percent ethanol and 5 percent glucose (highly acceptable). Both groups then had 5 percent ethanol in both tubes for 1 month and overindulged in the usual manner. The crucial evaluation was done by gradually increasing the glucose concentrations of one of the fluids over weeks from 0.7 percent to 5 percent glucose for both groups. This was to ascertain whether a distant history of having experienced a highly acceptable glucose solution would induce animals to give up the 5 percent ethanol overindulgence choice more readily than in the case of the group which had experienced only the rather dilute glucose, even though they were both given equivalent current choice opportunities. The answer was clear. As the glucose concentration was increased, animals with histories of 5 percent glucose gave up drinking 5 percent ethanol more readily than those with 0.7 percent glucose histories. The history of having experienced a highly preferred glucose solution militated against continuing to drink 5 percent ethanol, even when it was presently paired with a glucose concentration of relatively low acceptability. Perhaps a history of having experienced a potent reinforcing agent helps to ameliorate abusive overindulgence even when an attenuated version of the reinforcing agent becomes available.

Summary and Conclusions

An empirical biobehavioral research approach to the conditions generally identified as alcohol abuse and "alcoholism" suggests that the temporal ordering of biochemical, physiological, and behavioral events can provide an operational basis for characterizing the functional aspects of this complex disorder and for identifying distinguishable features of the alcohol abuse and dependence process. Within this interrelational framework, determinants are conceptualized and analyzed in the context of observed interactions among a host of continuous variables, both antecedent and consequent, with a focus on the conditions under which chronic and excessive alcohol drinking is generated and maintained, as well as modified therapeutically and prevented (*65,66*).

The available evidence suggests that "alcoholism" is a condition determined by a host of continuous variables rather than an entity possessing static qualities that imply intractability. The challenge for biobehavioral research is to determine how chronic and excessive alcohol drinking is generated, as well as the conditions under which such overindulgence can be attenuated and prevented. Recent experimental evidence has documented the fact that environmental context can dramatically alter the frequency and amount of drug taking even with the most abusable substances. Such contextual malleability is suggested as an important key to at least some inconsistencies in the literature with regard to the conditions under which chronic and excessive intake can be demonstrated in laboratory experimental models of alcoholism.

It has now been convincingly demonstrated, for example, that alcohol and a number of other substances taken both orally and intravenously will be self-administered excessively and chronically when even relatively minor constraints are imposed on the availability of an important commodity (for example, food) by providing only intermittent access to that commodity. The excessive alcohol intake under these conditions is concordant with other kinds of excessive behavior generated by the episodic delivery of a valued, deprival commodity in one domain that induces excessive behavior adjunctive to that domain. The adjunctive excess appears to depend, in the particular case of ethanol, upon the environmental opportunity to drink alcohol and the individual's history and capacity in this regard. These findings suggest that excessive and chronic alcohol ingestion can be viewed as a set of behaviors for which others might have substituted (and intermittently do) rather than as a highly specific disorder or disease.

Despite its venerable status among the theories of "alcoholism," physical dependence has long been relegated to a supporting role, albeit one of potential significance. As a result of research advances in an understanding of the factors generating and maintaining alcohol self-administration, physical dependence has now been deposed from its former stardom in this regard. No clear evidence exists that chronic and excessive alcohol intake is maintained by avoidance of the withdrawal syndrome associated with physiological dependence.

Although currently fashionable etiological and preventive orientations favor genetic influences as strong determinants of alcohol-related disorders, the main criteria for "alcoholism" present in populations are continuous distributions rather than discontinuous isolable entities. The alcohol consumption of a population is distributed unimodally rather than bimodally as a dominant genetic influence would require. And of course, troubled drinking is malleable, waxing and then entering periods of remission. Alcohol drinking, even in severely dependent individuals, remains susceptible to control by both antecedent and consequating environmental events. Perhaps most importantly from a biobehavioral research perspective, while laboratory animal models have demonstrated genetic determinants of alcohol preference, additional factors aside from genetic make-up and exposure to alcohol are clearly necessary to catalyze excessive, chronic intake. No genetic model has selectively been bred for chronic and excessive alcohol intake, per se.

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Research on Alcohol Problems from the Perspective of Changes—1940 to 1990

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Synopsis

The nature of alcohol problems, knowledge about alcohol use and abuse, and public perceptions and responses have all undergone substantial change during the past half-century. This paper traces some interrelationships between changes in alcohol-specific knowledge and behavior and other relevant social forces. The importance of change in the derivation, utilization, and interpretation of this knowledge is emphasized. Special emphasis is also placed on changes that make the integration of research between the biological and behavioral sciences desirable and necessary.

ALTHOUGH HISTORY is technically defined as a branch of knowledge that records and explains past events by enabling us to trace processes of change and their impact, history can contribute substantially to our understanding of the present and our projections for the future. This observation certainly is true for the field of alcohol studies in the United States. The theme of this paper is change. This encompasses changes in knowledge about alcohol and in beliefs and assumptions about what we know and can expect to learn in the future; changes in the social contexts of drinking and in who drinks what, where, when, with whom, and why; changes in attitudes and values about drinking and alcohol problems; changes in the classification and labeling of problems; changes in the risks and liabilities of intoxication; changes in our perceptions of the social costs of alcohol problems; and changes in social responses.

The time frame for this review is the half-century

'The question of chemical contamination is still broader when interactions with certain industrial compounds are included. Changes in the chemical environment in which drinking occurs constitute a significant but inadequately measured aspect of drinking behavior and problems.'

beginning in 1940. That was a time when national Prohibition had recently been repealed, the depression was ending, and the country was reluctantly preparing for war. Perhaps because of more pressing concerns and because many people were tired of issues associated with the Prohibition experiment, there was little national interest in alcohol problems. Alcoholics were heavily stigmatized, assumed to be of little worth, and usually blamed for their condition. Intoxication and its consequences was the primary focus of concern but, in reaction to Prohibition, there was a widespread reluctance to enforce anti-drunkenness controls.

At the scientific level, there was little ongoing research that related to alcohol problems. Investigators who did express an interest in the field seemed to derive the stigma that society applied to alcoholics. In academic circles there was a common assumption that first-rate scientists did not deal with such topics. There was virtually no support available from either private foundations or the Federal Government for research on any aspect of alcohol problems. However, at Yale University's Laboratory of Applied Physiology the nucleus was being formed for a multidisciplinary, multifunction center for studies on alcohol, and Howard W. Haggard, a physician and medical historian along with E. M. Jellinek, a biometrician, were preparing a book to be entitled "Alcohol Explored" (*1*) in which they would examine the current status of knowledge about alcohol, drinking, and alcohol problems. This book has provided a convenient reference for identifying some changes that have occurred since it appeared.

At the other end of our half-century continuum, we now have a massive expression of organized concerns and activities related to many aspects and issues of drinking behavior and alcohol problems. There are hundreds of books and numerous specialized journals that address particular aspects of alcohol problems. A Federal agency, the National Institute on Alcohol Abuse and Alcoholism, is supporting research and research training in many disciplines. A complex network of concerned organizations, both public and private, and Federal, State and local, is in place, providing programs of education, prevention, and treatment. Centers for alcohol-related research exist at many leading universities where highly respected scientists from a variety of disciplines are involved in the study of many alcohol-related questions and issues. A report recently issued by the Institute of Medicine, "Causes and Consequences of Alcohol Problems" (*2*), will provide a guide for measuring change at this other end of our half-century.

Examples of Change

While we are considering changes that have taken place in our knowledge about drinking and alcohol problems, it will be important to recognize that the phenomena being studied have themselves been changing and to identify and understand some of these changes, particularly as they impinge on each other.

Alcohol consumption. First, following World War II and until about 1980, the per capita consumption of alcohol in the United States rose steadily. Although the use of different criteria and measuring instruments precludes precise comparisons, it is reasonable to assume that this increase amounted to at least 30 percent. Since about 1980, however, there has been a modest, but apparently steady, decline in alcohol consumption. A constant throughout the period covered is the estimate that alcoholics and other chronic heavy consumers, comprising perhaps 10 percent of the drinking population, have accounted for about half of the alcohol consumed.

Problem drinking. As with the case of consumption, statistics on the numbers of problem drinkers have been subject to changing methods for deriving estimates and changing criteria. The statistics that we do have, however, suggest that the relative number of drinkers who encounter problems of one sort or another has also increased. It is impossible to measure changes in many types of problem drinking because of changes in a number of factors that have altered the criteria for defining and labeling problems. For example, there has emerged a much greater public consciousness about the consequences of both incidental intoxication and repeated alcohol abuse. At the same time, changes in the technology of our society have created a massive shift in the kinds of tasks that people are called upon to perform. This shift is away from energy-expending tasks, in which the energy-releasing properties of alcohol may have been functional, towards tasks requiring the exercise of motor control and placing other demands on brain function for which the depressant, intoxicating functions of alcohol are distinctly compromising. Thus, drinking behavior that was not considered a problem for those in energy-demanding occupations becomes defined as a problem for those in occupations that demand judgment, precise motor control, and rapid decisions. Consciousness of and concern about the role of alcohol in decreasing the efficiency and effectiveness of personnel has increased sharply among industrial and business employers and also within the military where there are now efforts to reverse long-held traditions that supported alcohol consumption.

In transportation, the increase in traffic density and in the power and speed of automobiles and comparable changes in boating and aviation have raised the relative liabilities of intoxication in vehicle operators. Associated with these changes there has emerged a distinctly greater public awareness of drinking and driving as a threat to public and personal safety.

Health and disease. In the arena of public health and medical care, several major changes have had an impact on public concerns about alcohol use and abuse. As part of a broad national movement for health promotion and disease prevention, alcohol abuse has been identified as a factor that significantly increases the risk for virtually all of the leading causes of death and disability. Also, as the costs of medical care and hospitalization have mounted, the role that alcohol abuse plays in conditions that require longer than usual periods of hospitalization and more costly intensive procedures has become a matter of national concern. Rising alarm about violent behavior, spouse and child abuse, crime, and risk taking that exposes others to danger has also focused public concern about the possible role of alcohol as a facilitating factor in behaviors that threaten the well-being of others.

It is significant that Haggard and Jellinek, while identifying most of the currently recognized alcohol-related diseases, specifically exonerated alcohol as a possible direct contributing cause. Instead they ascribed all such problems to nutritional deficiencies brought about because people who consume large amounts of alcohol acquire so much of their need for calories through the alcohol that they neglect to eat enough of the foods that provide essential vitamins, minerals, and other nutrients. It was their position that, other than hoarseness and gastrointestinal irritation, there were no significant direct toxic effects from alcohol experienced by the body.

They also dismissed as a myth possible associations between alcohol use by women and the production of damaged children. While they were speaking primarily of the centuries' old beliefs that alcohol use during procreation could affect offspring, they implicitly dismissed the probability of any toxic effects from alcohol on the fetus. In contrast, today it is assumed that alcohol has a direct role in the development of fetal alcohol effects, and numerous investigators are trying to identify specific mechanisms, critical amounts of alcohol that can cause fetal damage, and critical periods during pregnancy when damage may be induced.

Heredity. Another topic currently of great interest, but lightly dismissed by Haggard and Jellinek, is that of children of alcoholics. Noting that great interest had prevailed regarding hereditary liability as a cause of inebriety, they concluded that this belief was "entirely obsolete" (*1a*). After reviewing the literature on this subject, they concluded that at most 40 percent of inebriates could be said to have an "heredity taint," but that "the only permissible conclusion is that it is not a disposition toward inebriety which is inherited but rather a constitution which is so unstable that it does not offer sufficient resistance to the environmental risks of inebriety" (*1b*). In the 1980s, although the significance of environmental and behavioral factors is not excluded, it is assumed that certain genetically related traits are associated with both an individual's sensitivity responses to alcohol and risks for developing alcoholism or other drinking problems.

Chemical environment. Still another type of change that has altered both human responses to alcohol and the nature of alcohol problems for some users involves what may be called the chemical milieu in which alcohol use occurs. In 1940, when an individual became intoxicated, it was almost always due to alcohol use. There were some incidents of overdose involving the use of barbiturates and alcohol, but there were few other recognized, commonly occurring problems of adverse interactions between alcohol and other drugs. This picture has changed vastly in 50 years. There are now a large number of widely used medications that have a potential for undesirable or dangerous interactions with alcohol in a number of different ways. Of special concern are the anti-anxiety, anti-histamine, and sedative-inducing preparations that can interact synergistically or additively with alcohol to produce brain function compromises that far exceed what the user is accustomed to experience from alcohol alone. When marijuana and other illicit mood modifying drugs are added to this picture, the potential for complex chemical interactions with alcohol increases further. The question of chemical contamination is still broader when interactions with certain industrial compounds are included. Changes in the chemical environment in which drinking occurs constitute a significant but inadequately measured aspect of drinking behavior and drinking problems, and they also constitute a substantial challenge for future research. It is quite clear today that much intoxication is the result of multiple substance exposure and that many problems of alcoholism and other drug dependence are really cases of poly-addiction.

Biological Research

Of all the changes that we will identify, those that seem most dramatic from the viewpoint of knowledge advancement have occurred in the biological sciences. Here important research, much of it in the last 10 years, has substantially altered our perceptions about the relationships between alcohol and the human body. Research in genetics, biochemistry, physiology, and neuropharmacology has replaced rather simplistic notions about the actions of alcohol in the body. We now have much more complex and sophisticated explanations and descriptions of these mechanisms. Furthermore, current knowledge has permitted the formulation of new questions that, along with ever advancing technology, offer promise of even greater advances in our understanding of the actions of alcohol in the human body within the near future. A major impact of new knowledge has been to shift our perceptions about human responses to alcohol from a primary emphasis on homogeneity in response mechanisms to one of heterogeneity and the recognition that a substantial number of responses vary from person to person and, under different circumstances, within given persons. These variations include such factors as metabolic rates and processes; the nature, distribution, and roles of relevant enzymes; the sensitivity of target tissues; the action on membrane fluidity; the role and distribution of neurotransmitters; and many more. Much of what we have learned in recent years, and can expect to learn in the future, pertains to our understanding the relative sensitivity of individual persons to alcohol and the relative risks that individuals have of experiencing compromised task performance, intoxication, dependence of alcohol, alcoholism, or one or more alcohol-related diseases or social complications.

Of significance, along with new biological knowledge, is the growing recognition that the understanding of the actions of alcohol on the human body has limited meaning apart from knowledge about factors that determine whether people ever use alcohol and, if they do, what factors influence the quantity, frequency, and patterns of their exposure to alcohol. Put another way, biological knowledge can help us identify and understand ranges of relative individual sensitivities to alcohol and ranges of relative risk for experiencing one or more of the acute or chronic problems of alcohol. But such knowledge must be correlated with knowledge about factors that determine an individual's drinking experiences and cumulative exposure to alcohol.

For an understanding of drinking experiences and exposure to alcohol, we are dependent on the social and behavioral sciences. These disciplines are concerned with culturally influenced beliefs, attitudes, values, and customs; socially determined practices and pressures; psychologically related responses to environmental cues and the reinforcing significance of alcohol's mood modifying effects; and economic and political factors that influence relative ease of availability and access. Changing knowledge in the social and behavioral sciences has also been substantial in the period under review.

Social and Behavioral Research

In 1940, relatively little was known about the drinking practices of American society. Such data as were available were per capita consumption estimates derived from statistics reported by the distilling, brewing, and wine producing industries, augmented by reports on the number of illicit stills confiscated by the Internal Revenue Bureau. There were virtually no data on such questions as who in our diverse population were the drinkers and abstainers or such basic criteria as their ages, sex, ethnicity, occupations, education, income, religious preferences, places of residence, recreational patterns, and the like. Nor did we know anything about drinking practices such as what, how much, when, where, why, with whom, or in association with what activities or events people used alcohol. It was assumed, perhaps because of a post-prohibition paranoia, that most people considered their drinking practices very private and would not be willing to discuss them. Knowledge about drinking problems and problem drinkers was also sparse. This information was little more than what could be learned from the captive populations of mental hospitals and jails, and it provided a very limited and distorted picture of the characteristics of problem drinkers and the progression of their problems.

Today, in contrast, there is a vast amount of knowledge that addresses such fundamental questions as the patterns and conditions of human drinking behavior, the problems related to drinking, and societal responses to both drinking behavior and drinking problems. This change has come about in association with numerous factors including a change in public attitudes about drinking practices and problems, changing methodologies in such relevant disciplines as epidemiology, sociology, psychology, and anthropology, increased sources of funding for research and, as has occurred in the biological sciences, the involvement in research on alcohol-related questions of a number of extremely competent scientists. Research in the 1950s and 1960s accomplished much in revising long held assumptions and diminishing the impact of false stereotypes. These advances have been greatly refined and developed in the 1970s and 1980s.

As the Institute of Medicine report notes, current emphasis in social and epidemiologic research reflects a recognition of the significance of continuing change over time in drinking practices and drinking problems, for both individuals and populations, and in relation to variable settings and conditions. There is also a broadening frame of reference to incorporate studies of alcoholism within the context of alcohol-related beliefs, behavior, and problems (*2a*). As one example of current social and behavioral research, psychological studies have revealed important new insights about the significance of drinkers' beliefs and expectations with respect to alcohol and the reactions of others as these can influence certain of the effects experienced from drinking. Also of current interest are studies designed to identify the importance of social expectations or norms in influencing an individual's sense of need to drink or to drink to a point of intoxication. Such research is especially relevant in light of recent biological knowledge on individual variability in various responses to alcohol and capacity to drink safely. Also of note are epidemiologic studies that have provided new insights on the relationship between the prevalence of drinking problems in a population and factors controlling the availability of alcohol, such as cost.

> *'. . . psychological studies have revealed important new insights about the significance of drinkers' beliefs and expectations with respect to alcohol and the reactions of others as these can influence certain of the effects experienced from drinking. Also of current interest are studies designed to identify the importance of social expectations or norms in influencing an individual's sense of need to drink or to drink to a point of intoxication.'*

Summary

In this paper I have looked at several factors of change that appear to interact. These include changes in drinking practices and problems, changes in knowledge and in the pursuit of knowledge, and changes in society's response to drinking issues. The Institute of Medicine report emphasizes the importance of the phenomenon of change and the need for longitudinal and cross-sectional research (*2b*). Although Haggard and Jellinek did not explicitly address this issue, no one reading their book would expect them to have disagreed.

One point on which there is substantial explicit agreement between the 1940s perspective of Haggard and Jellinek and the perspective of the Institute of Medicine's report nearly 50 years later is the importance of integrating biological knowledge about alcohol and the human body with behavioral knowledge about factors that expose the body to alcohol. Haggard and Jellinek summarized biological knowledge rather briefly and devoted more attention to the need for more knowledge on such questions as what people drink and how much, why and how some people abuse rather than use alcohol, and what problems are associated with different levels of drinking. They repeatedly identified a need to know more about the behavioral and social factors that cause people to consume alcohol as a necessary corollary to knowledge about how alcohol affects the body once it is consumed.

The Institute of Medicine report, while summarizing a vast amount of biological knowledge, notes that "the many ways in which people respond to alcohol, their various capacities to drink safely, and the relative risks they run for the development of alcohol-related problems pose major questions for research. These are complex phenomena, occurring at the junction of biologic, behavioral, and social forces" (*2c*). The report also repeatedly stresses the need to integrate biological and behavioral approaches in the conceptualization and design of research.

In their recognition of the need for truly integrative biological and behavioral research, Haggard and Jellinek were clearly ahead of their time. Even though they established a multidisciplinary center of research at Yale in the 1940s, most of the research efforts there were parallel rather than integrated. The conceptual, ideological, and methodological barriers between disciplines were too strong to penetrate. Today, these barriers have already been diminished within the biological and within the social and behavioral sciences. It is often hard to distinguish between a biochemist and a microbiologist or between certain epidemiologists and sociologists. As we approach 1990, and the end of our half-century review of change in relation to alcohol problems in the United States, it seems clear that one goal for research on alcohol problems early in the next half-century must be a successful penetration of the biological-behavioral barrier.

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I certify that the statements made by me above are correct and complete.

Marian Priest Tebben
Executive Editor

NATIONAL CENTER FOR HEALTH STATISTICS DATA LINE

In this issue's Data Line column, the National Center for Health Statistics (NCHS) describes several of its surveys that provide data on the use of alcohol, the health consequences of alcohol use, and related data.

Although published reports represent the primary vehicle for disseminating NCHS data, these data are also available in public use data tapes. The data tapes permit researchers to cross-classify variables in different or greater detail than that generally presented in the NCHS publications.

A previously published report, "An Inventory of Alcohol, Drug, and Mental Health Data Available From the National Center for Health Statistics," describes the NCHS data on alcohol use in more detail than can be presented in this Data Line. For interested persons, current catalogs of NCHS publications and data tapes are available free of charge. Contact the Scientific and Technical Information Branch, 3700 East-West Highway, Rm. 1–57, Hyattsville, MD 20782, (301) 436-8500 for catalog copies or for further information on alcohol-related data.

The Surveys

The National Health Interview Survey (NHIS) is conducted annually and collects data through household interviews among a sample of the U.S. civilian population. The NHIS questionnaire contains two major parts. The first consists of topics that remain relatively the same from year to year. Information collected in this part of the survey provides data on the incidence of illness and injuries, prevalence of chronic diseases and impairments, disability, physician and dental visits, hospitalizations, and other health topics, as well as data on the demographic and socioeconomic characteristics of the population. The second part of the NHIS questionnaire contains special health topics added each year.

In 1977, the NHIS included a special health topic designed to obtain data on the prevalence of seven health practices, including the frequency and quantity of alcohol consumption. The questions on alcohol (as well as those on the other health practices) replicated, with some modification, those of the Alameda County study (*1*).

In 1983, the NHIS included a more extensive battery of questions on the use of alcohol. The 1983 NHIS contained questions on drinking practices, health practices, health conditions, self-classifications of drinking level, problems associated with drinking, and detailed items on the quantity and frequency of beer, wine, and liquor consumption.

In 1985, the NHIS added questions on health promotion and disease prevention devoted primarily to the collection of baseline data for monitoring progress toward specific 1990 objectives delineated in the 1980 Public Health Service report, "Promoting Health/Preventing Disease: Objectives for the Nation" (2). In addition to questions on the amount and frequency of alcohol use, the 1985 NHIS addressed the person's knowledge about the health effects of heavy alcohol drinking, for example, throat cancer, cirrhosis of the liver, and health problems of the newborn.

The 1988 NHIS gathered information on frequency and amount of alcohol consumption, reasons for quitting or abstaining, and alcohol use by the spouse and other relatives. Detailed information on alcohol behavior and associated problems were gathered separately for persons classified as lifetime infrequent drinkers, former drinkers, and current drinkers. The information collected in the 1988 NHIS battery of alcohol questions can be linked to the survey's data on child health, thereby greatly expanding the potential of research on the health-related repercussions of alcohol use.

The National Health and Nutrition Examination Survey (NHANES) collects data on the prevalence of specific conditions or chronic diseases, dental health, physiological and body measurements, and nutritional status and deficiencies. The data are collected by means of physician examinations, laboratory procedures, medical history, and standardized tests administered in mobile examination centers, as well as through interviews in the home.

In NHANES I (1971–75), questions on alcohol in the medical history questionnaire were asked directly of respondents ages 12 through 74. Information on the consumption of alcohol was obtained for the 24-hour period prior to the examination for all examinees ages 1 through 74 (either directly or through proxy response) in the dietary component of the survey.

In the dietary-frequency component of NHANES II (1976–80), data on alcohol use was gathered from all examinees ages 6 months through 74 years (either directly or by proxy response). Also in NHANES II, information on the consumption of alcohol was obtained for all examinees in the 24-hour dietary recall component of the survey.

The Hispanic HANES (HHANES) was a one-time survey conducted in 1982–84 of approximately 16,000 persons of Mexican American, Cuban American, and Puerto Rican descent. The HHANES contained a detailed battery of 73 questions relating to alcohol consumption. These questions were asked directly of all sample respondents aged 12–74 years old. Additionally, the HHANES contained a dietary-frequency component and a 24-hour recall component that followed the methodology of NHANES I and II.

NHANES I Epidemiologic Followup Study (NHEFS) is a collaborative project involving the NCHS, the National Institute on Aging, other components of the National Institutes of Health, and the Alcohol, Drug Abuse and Mental Health Administration. The study is a nationwide followup of 14,407 persons who were 25–74 years old when first examined in NHANES I (1971–75). The NHEFS investigates the relationships between clinical, nutritional, and behavioral factors assessed at baseline (NHANES I) and subsequent morbidity and mortality.

The NHEFS is being conducted in several waves. The initial followup phase of the study (1982–84) obtained data from personal interviews; measurements of pulse rate, weight, and blood pressure; data from hospital and nursing home records; and data from death certificates for decedents. Data on alcohol obtained in the first wave related to the reasons for abstaining, frequency of alcohol consumption, self-perception of quantity consumed, and adult history of drinking pattern.

A computer assisted telephone interview (CATI) followup of persons ages 55 years and older at baseline (3,980 subjects) was conducted in 1986 and a CATI followup of the entire cohort (11,750 subjects) was conducted in 1987. In 1986, the elderly were asked questions on the frequency of drinking during the previous year and the number of drinks of liquor, beer, and wine consumed during that period. This battery of questions was repeated for the full cohort in 1987.

Similar information on alcohol should become available from another wave of interviewing in 1990.

The National Hospital Discharge Survey (NHDS) is an annual survey of the use of non-Federal short-stay hospitals. Data on diagnoses, surgical procedures, and characteristics of inpatients (age, sex, and race) are collected along with information by size, location, and ownership of hospitals. Survey data are abstracted from a sample of medical records of approximately 500 participating hospitals.

In the NHDS, data on the inpatient treatment of persons involved with alcohol abuse are coded by the ICD–9–CM to show diagnoses such as "alcoholic psychoses," "alcohol dependence syndrome," "acute alcoholic intoxication," and "other and unspecified alcohol dependence" and "alcohol abuse." Beginning in 1980, alcohol data are also available coded under Diagnostic Related Groups (DRGs).

The National Nursing Home Survey (NNHS) has been conducted periodically since 1973–74. This survey is the source of data on the financial and other characteristics of nursing homes and also on their services and staff. Additionally, the NNHS provides data on the personal and health characteristics of nursing home patients.

In the NNHS, diagnostic information is collected from a sample of both current residents and discharged patients. Diagnostic data on "alcoholism" are available only from the 1977 and 1985 surveys.

In 1977, the NNHS collected diagnostic data using precoded check boxes. In this format, "alcoholism" was specifically identified as a primary diagnosis and as a chronic condition or impairment. In addition, "cirrhosis of the liver," a common sequalae of alcoholism, was also identified on the diagnostic checklist.

In the 1985 NNHS, diagnostic data were collected in an open-ended format and then coded to several categories of mental disorders, including alcoholic psychoses, alcohol dependence syndrome, and alcohol abuse. The 1985 NNHS also provided diagnostic data on cirrhosis of the liver.

Additionally, the 1985 NNHS Current Resident Questionnaire contained a checklist of mental disorders that included the category "alcohol abuse or dependence." (A similar checklist was not used for discharged patients.)

The National Natality and Fetal Mortality Survey is based on a sample of live birth and fetal death records. Data are obtained from questionnaires sent to the mothers and physicians, hospitals, and other medical care providers used by the mother.

One major area of investigation includes the effects of maternal drinking on infant health. In the 1980 cycle of this survey, married mothers were asked four questions related to drinking alcoholic beverages: (a) "Did you drink alcoholic beverages, that is, beer, wine, or liquor, during the 12 months before your 1980 delivery?" (*b*) "During the time you were pregnant, how often did you drink alcoholic beverages?" (c) "On the days that you drank, how many drinks did you have?" and (*d*) "On a typical day that you drank, what kinds of alcoholic beverages did you usually drink?"

The National Maternal and Infant Health Survey is being conducted in 1988. This survey has three components: live births, fetal deaths, and infant deaths. The alcohol questions in the 1988 survey have been modified somewhat but will produce data comparable to that obtained from the 1980 survey.

The National Mortality Followback Survey is designed to provide data on socioeconomic characteristics of deceased persons, use and payment for hospitals and institutional care during the patient's last year of life, and factors associated with health status. Questionnaires are sent to the decendents' next of kin and to the institutions that provided health care—hospitals, nursing homes, hospices, and other facilities. The survey was conducted annually from 1961 through 1968 and most recently in 1986.

In the 1986 survey, three alcohol-related questions were asked: (a) "In the person's entire adult life, did he or she have at least 12 drinks of any kind of alcoholic beverages, such as beer, wine, or liquor?" (*b*) "On the average, during adult life, how often did he or she drink any alcoholic beverages, such as beer, wine, or liquor?" and (*c*) "On the days that the person drank, how many drinks did he or she have on the average, per day?"

The National Survey of Family Growth (NSFG) is the source of data on family planning practices and attitudes, factors influencing trends and differences in fertility, and related aspects of maternal and child health.

The 1982–83 NSFG included a probability sample of approximately 8,000 women in their childbearing years (ages 15–44), regardless of their marital status. The survey had one alcohol-related question: "During your (last) pregnancy, how often (did/do) you usually drink alcoholic beverages, that is, beer, wine, or liquor?" The 1988 NSFG had a similar size and question on alcohol.

The Vital Registration System

Vital statistics. The 1989 revision of the U.S. Standard Certificate of Live Birth has a question on whether the mother used alcohol at any time during pregnancy and one on the average number of drinks she had per week. Since most States will be revising their birth certificates accordingly, these data on the mother's use of alcohol during pregnancy are expected to become available in 1991 for most of the 3.8 million births occurring annually in the United States.

Mortality statistics. Data on deaths related to or due to alcohol use are available for 100 percent of deaths registered in the United States each year. These data are obtained from death certificates and are coded in detail according to the ICD version in use at the time of data collection.

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2. Department of Health and Human Services: Promoting health/preventing disease: objectives for the nation. U.S. Government Printing Office, Washington, DC, 1980.

—PATRICIA GOLDEN, Special Assistant to the Division Director, Division of Epidemiology and Health Promotion; and ANITA POWELL, Health Statistician, Division of Epidemiology and Health Promotion.

Public Health Service Commissioned Corps to Celebrate Centennial

The U.S. Public Health Service (PHS) Commissioned Corps will celebrate its 100th anniversary on January 4, 1989.

The Centennial theme will be the contributions of the Commissioned Corps to PHS and the nation's health, reflecting its current revitalized status and mission. The Commission Corps is one of the uniformed services, and has 6,000 members serving worldwide.

Centennial organizers are encouraging the participation of active duty and retired officers, together with the public health community at large and the entire PHS. Activities are planned for the Washington, DC, area and wherever Corps officers are stationed.

Activities now planned to begin the Centennial include:

- The Centennial Convocation, which will deal with historical aspects of the Corps and its impact on the public health of the nation.
- The Centennial Gala, a banquet celebration, with music by the U.S. Coast Guard Band.
- Professional and educational activities arranged by PHS and other agencies and organizations.

Activities scheduled throughout the Centennial year include conferences and symposia sponsored by Federal health agencies whose staffs include Commissioned Corps members, as well as by other professional groups and organizations. Related Centennial activities planned are:

A U.S. Postal Service commemorative postal cachet and cancellation on the Centennial date

A Presidential Proclamation for the anniversary date and State Governor proclamations in States where Corps officers are on duty

A project to document historical information about PHS and its relationship with the Commissioned Corps from its inception, to be contained in a book to be published as part of the observances

Oral histories from those who have served as Surgeon General and in other key posts

Special exhibits at PHS installations and specialized medical and health museums

A poster reproduction of the historical events of PHS and the Corps

A commemorative medallion or coin to be designed, struck, and issued for the occasion

Special lyrics and a recording of the "PHS March," which is the only march copyrighted by a uniformed service.

Recruitment and special activities for the Commissioned Officer Student Training and Externship Program (COSTEP). Information on COSTEP is available by calling 1-800/221-9393, 703/734-6855 in Virginia.

A master program of all events for the year will be mailed to all active duty officers. Additional information on the Centennial is available from PHS Commissioned Corps Centennial, P.O. Box 30100, Bethesda, MD 20814.

—CDR. STEVEN R. MOORE, Office of the Surgeon General, Public Health Service, Parklawn Building, Room 7-05, Rockville, MD 20857.

Call for Papers: 1989 Public Health Conference on Records and Statistics

The 22d biennial Public Health Conference on Records and Statistics will be held in Washington, DC, July 7-19, 1989. The conference, which serves as a national forum for the latest advances in public health statistics, is sponsored by the National Center for Health Statistics (NCHS).

The theme of the 1989 conference is "Challenges for Public Health Statistics in the 1990s." Three major areas will be addressed: promotion and prevention, surveillance and epidemiology, and targeting services. Presentations will be organized in sessions focusing on policies, programs, and services; methodological and analytical issues; and trends, projections, and goals.

Invited and contributed papers will be featured. Abstracts of papers to be considered must be received by NCHS by January 13. For more information, contact Nancy Hamilton, OPEP, PHCRS, NCHS, 3700 East-West Highway, Rm. 2-12, Hyattsville, MD 20782. Telephone: (301) 436-7122.

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Key to Classification of Subject Index

a	Original article
br	Brief report
e	Editorial
ltr	Letter

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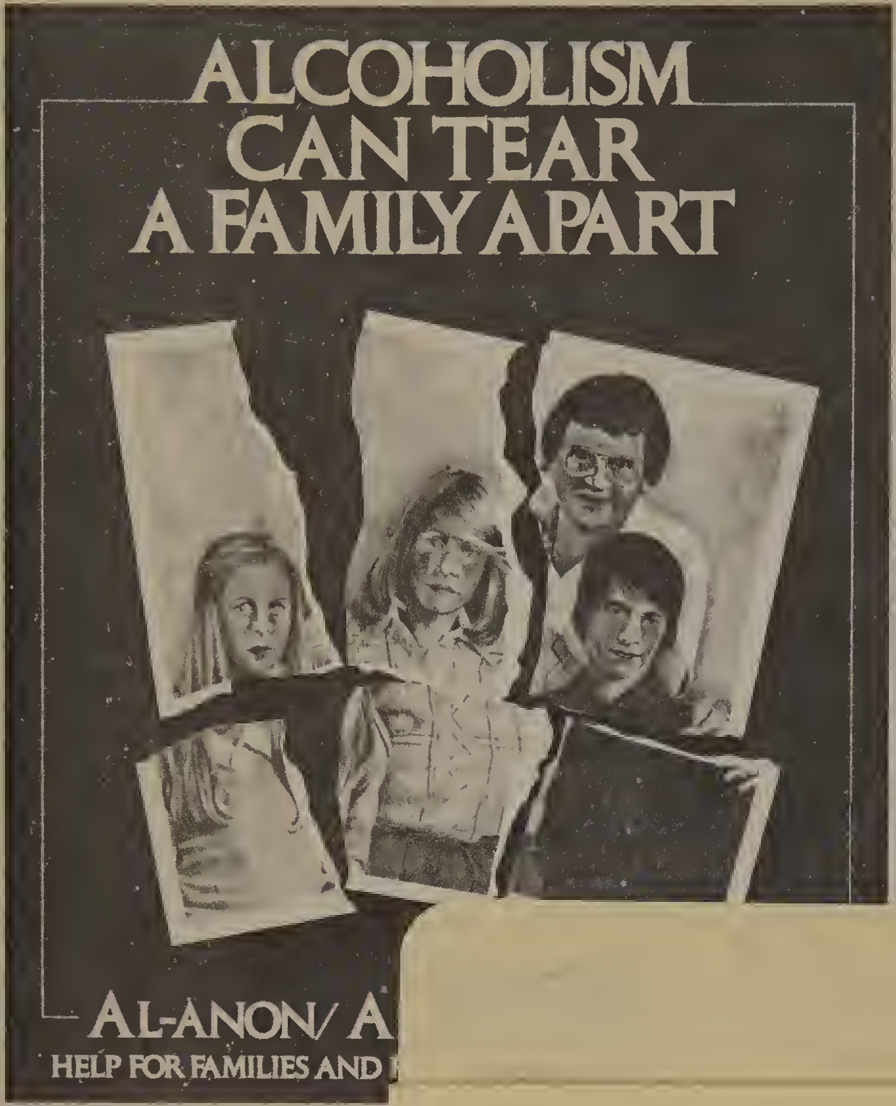
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Weekly Reader

ALCOHOL

A Drug That Can Harm the Body

Alcohol is a drug. It can be dangerous. It can be habit-forming. It can harm health. Here's what alcohol can do to different parts of the body.

BRAIN

Alcohol may reduce the flow of oxygen to the brain. Fuzzy thinking, staggering, and confused speech can result. With increased amounts, alcohol can cause brain damage, unconsciousness, or death. Alcohol even in small amounts makes it difficult to get a good night's sleep.

EYES

Alcohol relaxes eye muscles, making it difficult to focus on anything. The result can be blurred vision. In large amounts, alcohol can reduce the ability to see to the right and left.

NOSE, TONGUE

Alcohol, even in small amounts, can reduce the ability to smell and taste.

HEART

Alcohol can increase the workload of the heart. Heavy use of alcohol over a period of years can damage the main heart muscle. Alcohol can also cause the heart to beat irregularly. An irregular heartbeat can lead to heart failure in some cases. Alcohol can raise blood pressure.

LIVER

Alcohol over a period of time can cause the liver to become swollen and tender. This can keep the liver from working properly and bring on diseases that can kill. An alcohol-damaged liver can also reduce the production of substances that clot blood and help stop bleeding.

STOMACH

Alcohol irritates the lining of the stomach and can cause vomiting. Repeated use can sometimes cause sores called ulcers. Alcohol can lead to cancer of the esophagus, the tube that takes food to the stomach.

PANCREAS

Alcohol can cause the lining of the pancreas to swell. The swelling can block off the passage to the small intestine. Chemicals needed in the small intestine to aid digestion cannot get through. The blocked chemicals begin to digest the pancreas itself, causing pain, vomiting, and possibly death.

KIDNEYS

Alcohol can stop the kidneys from keeping the right balance of fluid in the body. Too much fluid in the body can cause a rise in blood pressure.

VEINS, ARTERIES

Alcohol widens blood vessels near the surface of the skin, causing a loss of heat from the body. Swollen arteries in the head can put pressure on nerves and cause headaches.

BLOOD

Alcohol reduces the ability of the bone marrow to make red blood cells, causing a condition called anemia. A person with anemia feels weak and tired. Alcohol also reduces the number of white blood cells and their ability to fight infection. Alcohol interferes with normal blood clotting.

SKIN

Alcohol seems to worsen skin diseases such as acne.

MUSCLES

Alcohol may lead to muscle weakness and muscle destruction.

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